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Skin and venereal diseases

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КОЖНЫЕ
И ВЕНЕРИЧЕСКИЕ
БОЛЕЗНИ

SKIN AND VENEREAL DISEASES

Textbook for Secondary Medical Schools

by

L. FANDEYEV
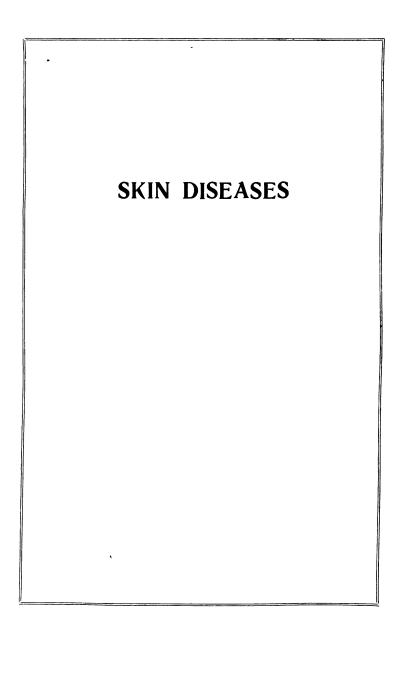
Translated from the Russian
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SKIN DISEASES

INTRODUCTION

Dermatology is the science of the skin and skin diseases; venereology is the study of venereal diseases.

Skin and venereal diseases began to be studied in antiquity. Descriptions of signs of various skin and venereal diseases and methods of their treatment can be found in manuscripts of ancient China, India, Egypt, Greece and other countries.

The development of dermatology and venereology as scientific disciplines dates from the second half of the 19th century. Russian dermatology and venereology came into existence at the same time.

Two trends in the study of skin diseases took shape in Western Europe in the middle of the 19th century; these trends were headed by the German and French dermatological schools. The adherents of the German dermatological school attached great importance to studying eruptions on the skin and their subsequent transformations, as well as to pathoanatomical changes occurring in skin diseases. They devoted considerable attention to the role played by various extraneous stimuli in the origin of skin diseases. The studies of these factors contributed to the development of the science of skin and venereal diseases. At the same time some representatives of the German dermatological school underestimated the significance of the general condition of the organism and the role of nervous and visceral dysfunction in the development of skin diseases; they often regarded skin diseases as purely local processes. This explains their attempts to treat patients suffering from skin diseases mainly with external agents and their underestimation of methods of general treatment.

The French dermatological school attached the greatest importance in the origin of skin diseases to changes within the organism, and this was its positive aspect. However, the ideas about these changes were often very vague. The French dermatologists considered many cases of skin diseases to be the result of "spoilage of juices". The scientists of the French school often gave too little thought to the external environment. The French dermatological school has made a valuable contribution to the study of fungus infections of the skin and to the elaboration of the methods of their treatment.

The development of the sciences of skin and venereal diseases was enormously influenced by progressive Russian medical scien-

ends, the fathers of Russian scientific medicine—M. Mudrov, S. Botkin, I. Sechenov and G. Zakhariyn.

The views of these scientists are characterised by their recognition of the leading role of the nervous system in the vital activities of the organism and their strivings to treat, "not the disease, but the patient".

The fathers of Russian dermatology and venereology were A. Polotebnov, A. Pospelov and V. Tarnovsky.

Russian dermatology and venereology are noted, on the one hand, for their view of skin diseases as diseases of the entire organism and, on the other hand, for their striving to improve medical aid to the patients suffering from skin and venereal diseases and to control their spread.

Soviet dermatology and venereology aim, not only at treating skin and venereal diseases, but also and mainly at preventing them. This *prophylactic* trend is the distinguishing characteristic of medical science of the Socialist state, amelioration of the health of the working people being one of its most important objectives.

Soviet dermatology and venereology derive their strength from their close contact with practice. The achievements of these sciences are systematically introduced into the practical work of hospitals and clinics for skin and venereal diseases. At the same time the practical experience of these hospitals and clinics is carefully generalised and studied in research institutions. Medical practitioners are widely enlisted for participation in scientific work.

ANATOMY AND PHYSIOLOGY OF THE SKIN

The skin is a natural covering and inseparable part of the human body. By separating the organism from the external environment it performs the important function of protecting the organism from unfavourable influences of the environment. It also participates in a number of very important processes, namely, thermoregulation, metabolism and excretion of waste products.

Structure of the Skin

The anatomical structure of the skin fits it for the performance of these important functions.

The total surface of the skin reaches 1.6 m². In adults the skin weighs about 18 per cent of their body weight. It has superficial furrows and deeper folds.

The superficial furrows cover the entire skin and by crossing form *skin fields* in the shape of triangles and rhombuses. On healthy skin the pattern of the skin fields is very delicate. It is more clearly marked on the dorsal surfaces of the hands and wrists. On the palms and soles, as well as on the palmar surfaces of the fingers and plantar surfaces of the toes the furrows are somewhat deeper and for the most part run in parallels.

The skin is composed of three layers: (1) epidermis or external layer, (2) true skin or derma, and (3) subcutaneous adipose layer or panniculus adiposus.
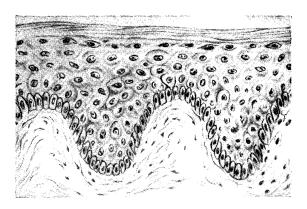

Fig. 1. Structure of the epidermis (from P. Grigoriev)

The **epidermis** consists of epithelial cells which possess great ability to multiply and replace the destroyed cells of this layer. Owing to this ability any wounds suffered by the skin, as a result of injury or skin disease, heal quickly and without leaving a trace.

Microscopic examination of the epidermis shows that it is composed of five layers: (1) stratum germinativum or basale, (2) prickle-cell layer, (3) stratum granulosum, (4) stratum lucidum, and (5) stratum corneum (Fig. 1).

The *stratum germinativum* or basale adheres to the true skin or derma. It consists of one layer of cylindrical cells with large and easily stained nuclei. The cells of the stratum germinativum do not adhere to each other, but are divided by narrow fissures. These fissures called intercellular canaliculi extend into similar canaliculi of the overlying prickle-cell layer of the epidermis. Lymph from the lymphatic fissures of the derma penetrates into the canaliculi of the epidermis and circulates through them. The cells of the stratum basale are interconnected by protoplasmic bridges. The epidermis has no blood vessels, and the lymph entering the intercellular canaliculi brings nutrient substances into the epidermis and removes the metabolites.

The protoplasm of the cells of the stratum germinativum has grains of melanin (pigment) which are coloured from light brown to dark brown.

In dark-complexioned people the cells of the stratum germinativum contain more pigment than do those of light-complexioned people. The stratum germinativum in the skin of people of tropical countries contains still more grains of pigment. Sun-tan is due to an increase in the amount of pigment in the stratum germinativum. The increased deposition of pigment in response to insolation is a defense reaction of the organism; by covering the nucleus lumps of pigment protect from the harmful effects of ultraviolet rays not only the nucleus, but also the deeper cells. Among the cells of the stratum basale there are also special pigment cells— melanoblasts and melanocytes.

The cells of the epidermis multiply in the stratum germinativum. The young cells formed as the result of division replace the older cells and crowd them into the prickle-cell layer. No multiplication of cells is normally observed in the prickle-cell and other overlying layers of the epidermis.

The *prickle-cell layer* is made up of an average of 4-6 rows of cells. The interpapillary prominences of the epidermis have more rows of cells (Fig. 2). The prickle cells are large, polyhedral and have large, light nuclei. These cells also have numerous intercellular protoplasmic bridges and are separated from each other by intercellular canaliculi.

The *stratum granulosum* is composed of 1-3 rows of elongated cells arranged parallel to the surface of the skin. These cells have
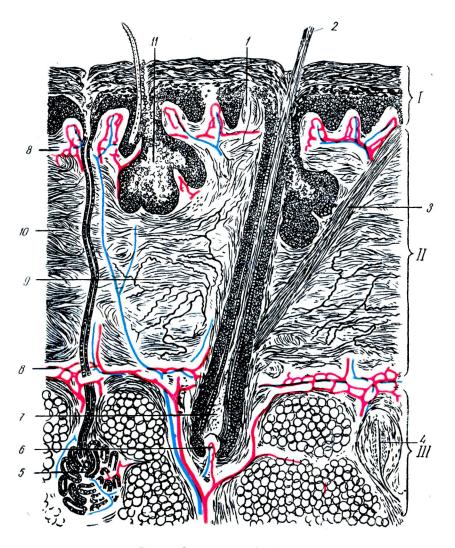
Fig. 2. Structure of the skin

I—epidermis; II—true skin; III—panniculus adiposus; 1—Meissner's corpuscle; 2—hair; 3—hair-erecting muscle; 4—Vater-Pacini corpuscle; 5—sweat gland; 6—hair papilla; 7—hair bulb; 8—blood vessels; 9—elastic fibres; 10—collagenous fibres; 11—sebaceous gland
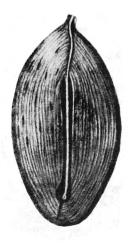
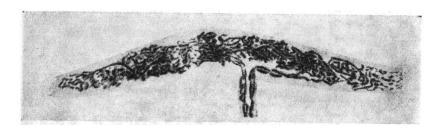Fig. 3. Nerve endings in the skin.
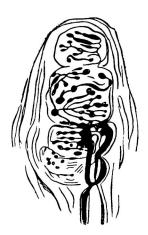Vater-Pacini corpuscle

Fig. 4. Ruffini's corpuscle

Fig. 5. Meissner's corpuscle

Fig. 6. Krause's corpuscle
(from V. Nikolsky)

pale nuclei. The protoplasm contains numerous grains of *kerato-hyalin*, an albuminous substance which is an early phase of the formation of keratin—the horny substance of the skin.

The three lower layers of the epidermis—stratum germinativum, prickle-cell layer and stratum granulosum—are often designated together as the malpighian layer.

Over the stratum granulosum is the *stratum lucidum*. Under the microscope this layer appears as a shiny thin band; it is composed of 1-2 rows of flat, shiny cells without nuclei. The protoplasm of these cells contains *eleidin* (an albuminous substance). Eleidin is a product of further transformation of keratohyalin into a horny substance.

The *stratum corneum* is the outermost layer of the epidermis. It is formed of several intimately united rows of flat, thin, horny plates overlying each other.

The horny plates are composed of completely keratinised cells of the epidermis without nuclei. Their protoplasm has complete-ly changed to keratin, the end product of the process of keratin-isation. The stratum corneum is the thickest on the palms and the soles of the feet. On the surface of the stratum corneum the horny plates are less adherent and are gradually cast off; this is known as desquamation which is a normal physiologic process.

True skin or derma. The second layer of the skin—the true skin or derma—is located under the epidermis (see Fig. 2). The derma abounds in connective tissue fibres which form interweav-ing bundles. The connective tissue of the derma contains but few cells.

Two basic types of connective tissue fibres—collagenous and elastic—are distinguished. The interspaces between the bundles of fibres are filled with the basic amorphous substance which plays a very important part in the processes of metabolism and in the protective functions of the skin.

There is also a third type of connective tissue fibres—reticular fibres—which are arranged in a thin layer between the epidermis and the derma, and around sebaceous and sweat glands, hair follicles and muscles of the skin.

At the junction with the epidermis the derma forms an undu-late surface, its papillae projecting into the epidermis. The junction between the epidermis and the derma is very clearly defined.

The derma is composed of two layers: papillary and reticular. The papillary layer is next under the epidermis. The bundles of connective tissue fibres in the papillary layer are quite delicate and interweave in various directions. Many bundles run perpendic-ularly to the surface of the skin and project into the papillae.

The reticular layer consists of thicker bundles of fibres which by interweaving form a dense network. A large part of these bundles is arranged parallel to the surface of the skin. This structure

of the derma made up of interweaving bundles of connective tissue fibres imparts great strength and elasticity to it. The thickness of the derma ranges in the different parts of the skin from 0.5 to 4 mm. The derma has no clearly defined junction with the underlying panniculus adiposus.

The **panniculus adiposus** (subcutaneous adipose layer) also consists of interweaving loose bundles of connective tissue fibres which form a large-mesh network. The meshes of this network contain fat lobules—accumulations of fat cells.

The panniculus adiposus is thicker on the abdomen, hips and buttocks. It is attached to the underlying fascia by connective tissue bundles.

Blood and lymph vessels of the skin. The skin has a well developed system of blood and lymph vessels. The blood vessels of the skin may hold up to one-fifth of the total mass of blood of the human organism. In the processes of blood circulation the skin performs the function of an important blood depot.

The arterial trunks enter the panniculus adiposus from deeper tissues. Here they give off branches which supply the panniculus adiposus and at the junction with the derma form an arterial plexus known as the *deep cutaneous vascular network*.

The deep network gives off vessels which ascend to the derma. These vessels give rise to arterial branches which supply the derma, as well as the sebaceous and sweat glands, hair, muscles and nerve endings located in the derma. Another arterial plexus— the *superficial cutaneous vascular network*—is formed at the boundary between the reticular and papillary layers. This network gives off an arteriole into each individual papilla. The terminal arterial branches of the skin break up into capillaries which supply the tissues of the skin. The capillaries gradually coalesce and give rise to the veins of the skin. The venous vessels of the skin run parallel to the arterial vessels.

The lymphatic system of the skin begins with the intercellular spaces of the epidermis and the numerous lymph spaces of the derma. The lymph vessels run alongside the blood vessels and form a superficial and deep vascular network.

The blood vessels of the skin have the ability rapidly to alter their lumens, i.e., to dilate or become constricted reflexly in response to stimulation of the nerve endings by heat, cold, mechanical factors and chemical substances. Reflex dilatation or constriction of the blood vessels may also result from various emotions—joy, fear, rage, etc.

Nerves of the skin. The human skin abounds in nerve fibres and endings.

The nerve trunks entering the skin form a nervous plexus in the panniculus adiposus. This plexus gives off numerous small trunks which run to the derma where they form new plexuses.

The nervous plexuses of the panniculus adiposus and derma give off branches to the hair follicles, sebaceous and sweat glands, muscles and vessels. Moreover, the skin has nerve fibres which run alongside the blood vessels. The skin contains an especially large number of sensory nerves which terminate either in free nerve endings (see Fig. 7) or in special end organs.

Most of the free nerve endings are in the epidermis; the derma has the fewest nerve endings which are believed to be sensitive to pain. The end organs have a peculiar and complex structure (Figs. 3, 4, 5, 6 and 7).

It is held that this difference in the end organs indicates a specialised sensitivity of each type of nerve ending to a particular form of stimulation—mechanical, heat, cold. Most of the nerve endings of all types are in the derma and in the lower layers of the epidermis.

Fig. 7. Free nerve endings in the epidermis (from M. Zheltakov)

Muscles of the skin. The skin contains mainly smooth, i.e., involuntary, muscles; these are primarily the muscles which raise the hair (see Fig. 2). On contracting they erect the hair. These muscles contract reflexly in response to stimulation of the skin by mechanical factors or by cold ("goose flesh"), or under the influence of fear, rage and other emotions (the hair "stands on end").

Smooth muscle fibres are also found in the walls of vessels and in sweat glands.

Striated or voluntary muscles are found only in the skin of the face. They are known as mimetic muscles because their contraction imparts mobility and expression to the face, which thus reflects the changes in man's emotions.

Sweat and sebaceous glands. Structurally the sweat glands are plain tubular glands. Each sweat gland consists of a body and duct. The body of the sweat gland, i.e., its secretory part, is in the subcutaneous layer or in the lower part of the derma. It looks like a coiled tube consisting of one row of glandular cells and a membrane. The membrane is made up of connective tissue and smooth muscle fibres.

The sweat glands are abundantly supplied with blood vessels and nerve fibres, and secrete perspiration. The skin of the soft parts of the fingers and toes, the palms and soles of the feet, the axillary and inguinal regions have the most sweat glands. There

are no sweat glands on the vermilion border, the glans penis and the inner leaf of the prepuce.

In addition to the usual, so-called *eccrine*, glands man also has larger, *apocrine* glands. They attain full development at the time of sexual maturation; their function is connected with that of the gonads. The apocrine glands are found mainly in the axillary, mammary, anal and genital areas. The ducts of the eccrine glands open on the surface of the skin, while those of the apocrine glands enter the upper part of the hair follicles.

The sebaceous glands have an alveolar or saccular structure and are found in the derma. The ducts of most of these glands enter the upper third of the hair follicles. Only a small number of sebaceous glands open directly on the surface of the skin (inner-leaf of the prepuce, labia minora, eyelids, nipples and lips). The secretion of the sebaceous glands—sebum—is intended for lubrication of the skin. It is formed by fatty degeneration of the epithelial cells of the glands.

Most of the sebaceous glands are found on the head, face and upper part of the back, and are absent on the palms and the soles of the feet. They attain their greatest development at the time of sexual maturation.

Hair. There is some hair on most of the skin and no hair at all on the skin of the palms, soles of the feet, the dorsal surface of the terminal phalanges of the fingers and toes, the vermilion border, the nipples, labia minora, inner aspect of the labia majora, glans penis and inner leaf of the prepuce.

The following types of hair are distinguished: (1) long hair (on the head, beard and mustache, in the axillae, and on the pubes and the genitals); (2) bristles (eyebrows, eyelashes, in the external auditory meatus and in the nares); (3) downy hair found on the other parts of the skin. A hair consists of a free (external) part or shaft which rises above the surface of the skin, and a root which is concealed in the skin. The root is found in a hair sac (hair follicle). The lower end of the hair roots is expanded into a hair bulb (see Fig. 2) which is the site of multiplication of the cells forming the hair. At the bottom the hair bulb is adjoined by a *hair papilla* which contains many blood vessels and nerve fibres required for the nourishment and growth of the hair. A duct of one or several sebaceous glands opens into the upper third of each hair follicle. On contraction of the muscle which raises the hair the latter straightens out and the secretion of the sebaceous glands is squeezed out of the hair follicle.

Nails are dense, quadrangular horny plates covering the dorsal aspect of the terminal phalanges of the fingers and toes. They rest on so-called nail beds and their function is to protect the terminal phalanges from mechanical injuries. The anterior edge of a nail is free, its posterior and lateral edges are surrounded by a

skin fold and project into it. The upper part of the skin fold covers the nail and is known as the *nail fold.*

A nail consists of a nail body and a nail root. The nail root is the posterior part of the nail found in the posterior skin fold and covered by the proximal nail fold. Only a small part of the nail root projects from under the nail fold as a whitish semilunar area (lunula unguis). The lunula unguis can be seen best on the thumb nails. The nail root rests on the posterior part of the nail bed and is called the *matrix.* The matrix is the site where the nail is formed.

The hair, nails and the sebaceous and sweat glands are called *cutaneous appendages.*

Pigment formation. The cells of the stratum basale of the epidermis contain grains of *melanin.* They are also found in the pigment cells of the papillary layer of the derma, which are called melanoblasts.

PHYSIOLOGY OF THE SKIN

The skin is intimately connected with the central nervous system and through it with the other organs and systems.

The skin plays an important part in the metabolic processes which are regulated by the central nervous system. It also plays a significant part in water and mineral metabolism. It is one of the most important water depots of the organism capable of retaining water and large amounts of mineral salts.

The skin participates in the processes of protein, fat and carbohydrate metabolism. Keratinisation of the epidermis is associated with complex transformations of albuminous substances and formation of keratohyalin, then eleidin and, lastly, keratin. The skin is one of the principal fat depots of the organism. The amount of fat deposited in the fat lobules of the panniculus adiposus increases or decreases, depending on the processes of fat metabolism. The skin also plays a significant part in the metabolism of vitamins A, C and D, and the vitamin B complex (B_1, B_2 and PP).

The skin is a *sense organ.* The numerous complex nervous receptors in the skin, connected through nerve fibres with the central nervous system, serve the skin to exercise its function of a sense organ. The "specialised" receptors of the skin receive various stimuli from the external environment and transmit the stimulation to the central nervous system.

The nervous receptors of the skin perceive the continuous changes in the external environment, i.e., variations in the temperature and humidity, and the various contacts of the human body with the surrounding objects. The "signals" transmitted from the receptors of the skin to the central nervous system are transformed in the cerebral cortex into sensations of heat, cold,

pain, itching, contact, pressure, etc. In other words, the skin together with the other sense organs (vision, hearing, touch and taste) enables man correctly to orient himself in his surroundings. In the process of evolution of the human organism the tactile ability of the skin developed to a high degree. Especially sensitive is the skin of the fingers and lips.

Protective properties of the skin. As a natural covering of the body the skin protects the organism from various unfavourable external influences—physical, chemical and infectious. The physical influences on the skin are mechanical, thermal (heat and cold), electrical and actinic (the sun, ultra-violet lamps, roentgen rays). The skin very well resists various mechanical influences—friction, shock, pressure, stretching. The skin owes its strength to its structure—the density and flexibility of the stratum corneum, the resilient, elastic network of connective tissue fibres of the derma and panniculus adiposus, and the shock-absorbing properties of the loose and resilient fatty lining of the panniculus adiposus. As a poor heat conductor the skin protects the human organism from overheating and cooling in cases of variations in the external temperature and thereby helps to maintain a constant body temperature.

The impermeability of the stratum corneum of the skin to most chemical substances and water and the presence of a fatty lubrication on its surface protect the skin and the deeper tissues from the harmful influences of many chemical substances and from maceration by water and moist air. The numerous receptors of the skin also play a protective role by signalling to the central nervous system every contact with the skin, every pain and temperature stimulus. This enables the organism to respond to any influence that threatens to injure the skin and the deeper tissues. In some cases the response is an unconditioned reflex, for example, an instant jerking away of the hand upon its contact with a hot or a stabbing object; in other cases it is a conscious act. For example, in response to the sensation of something crawling on the surface of the skin man examines the given area or shakes off the insect responsible for the sensation of the skin.

Intact skin protects the organism from penetration of various infectious agents. A healthy skin is impermeable to most pathogenic microbes coming in contact with its surface. The immune properties of the skin also prevent pathogenic microbes from penetrating into the skin and developing therein. The main substance of the derma possesses antimicrobial action. Other protective substances of the type of antibodies have also been discovered in the skin.

Participation of the skin in heat-regulating processes. As the organ directly in touch with the external environment the skin plays an important part in the heat-regulating processes.

The organism loses heat into the external environment largely through the skin by radiation, heat conduction and evaporation of sweat. If the conditions within the organism lead to heat production in excess of heat loss, the heat loss increases as a result of reflex "orders" issued by the central nervous system. This is achieved, firstly, by reflex dilation of the cutaneous blood vessels. The amount of blood circulating in the vascular network of the skin increases by the delivery of warmer blood from the internal organs. Consequently, the heat loss increases by radiation and conduction. Secondly, the heat loss increases by a reflex increase in perspiration. Upon the increase in perspiration the expenditure of heat on evaporation of the secreted sweat also increases. When the heat loss is greater than heat production, the central nervous system sends reflex impulses which cause a decrease in heat loss. In such cases the heat loss diminishes as a result of constriction of the cutaneous blood vessels, a flow of blood to the internal organs and a decrease in perspiration.

Secretory function of the skin. The secretory function of the skin is performed by the sweat and sebaceous glands.

The sweat glands are a constituent part of man's excretory system. Sweat is a fluid with a low specific gravity (about 1004) and a composition somewhat similar to that of urine. It is 98 per cent water and 2 per cent solid residue. In addition to water the organism excretes through the sweat glands salts (sodium chloride, etc.) and protein metabolites (urea, uric acid, ammonia, etc.). Moreover, the secretion of sweat is associated with processes of heat regulation. Close to 500-600 ml of sweat is excreted in 24 hours. In cases of hard physical work, high external temperature and fever the amount of excreted sweat may sharply increase (2-4 litres and even more).

The sebaceous glands secrete sebum which serves as an oily lubricant of the skin. About 20 g of sebum is secreted in 24 hours. The secretion of the sebaceous glands plays an important part in preserving the integrity of the stratum corneum and its impermeability to water, chemical substances and most microorganisms. The fatty lubrication softens the skin, imparts elasticity to the stratum corneum and facilitates the friction of contacting surfaces of the skin.

The acid reaction of the sebum hinders the development of most of the pathogenic microorganisms occurring on the surface of the skin. Sebaceous glands also participate in the excretory functions of the organism. Certain metabolites are eliminated in the sebum.

Absorptive ability of the skin. Healthy, undamaged skin is scarcely able to absorb water, other liquids and solids. Volatile fluids—chloroform, ether, etc.—are absorbed more readily. Solids can be absorbed if they are dissolved in volatile fluids. Absorption

of solids is favoured by their vigorous inunction into the skin. Maceration, i.e., softening (by steeping), of the epidermis, mechanical injuries to and inflammatory processes in the skin sharply increase the ability of the skin to absorb various liquids and solids. The permeability of the normal skin to oxygen, carbon dioxide and water vapours enables the skin to participate in the respiratory function of the organism which consists in absorption of oxygen and elimination of carbon dioxide and water vapours. Thus the skin supplements, as it were, the basic respiratory function performed by the lungs.

Age and sex characteristics of the skin. The structure and function of the skin have certain characteristics associated with age and sex.

The skin of children, especially of infants, contains much more water than that of adults. It is also thinner, more delicate, more vulnerable and more readily macerated than the skin of adults. The sebaceous and hair follicles of children are underdeveloped, their development being completed at the time of sexual maturation.

In old age the skin undergoes atrophic changes and the secretion of the sebaceous glands diminishes, which is conducive to dryness of the skin. The atrophic changes in the derma result in a thinning of the skin and loss of its normal elasticity. Senile skin contains more water. The regenerative ability of the skin, i.e., replacement of the dead or destroyed cells, appreciably weakens.

In most cases women's skin is thinner and more delicate. Women usually have less hair on the skin of their extremities and trunk than do men. In women the subcutaneous adipose layer is usually more developed, especially on the hips, buttocks and abdomen, than it is in men.

HYGIENE OF THE SKIN

Preservation of the normal condition of the skin and its proper functioning requires rational care.

Cleanliness is the most important condition for keeping the skin healthy. The surface of the skin is continuously soiled with sweat and sebum. The cast-off cells of the stratum corneum accumulate on the surface of the skin. The dust from the surrounding air also settles there. All this may be retained on the skin and by putrefying may offer a good nutrient medium to various microbes occurring on the surface of the skin.

How often must we wash the skin? This depends primarily on how fast and to what extent the skin is soiled. The exposed parts of the body (face, neck and hands), skin folds, and parts of the skin which come in contact with urine and excrements are the ones most readily soiled. People with a healthy skin must

wash the face and neck once or twice a day. The axillary, inguinal, and anal areas, as well as the external genitals and feet, must be washed with soap and water once a day. The whole body must be washed in a bathtub or given a shower bath once a week. However, people working where there is a good deal of dust, where the skin easily becomes soiled or where they perspire freely must wash the skin more often—as often as it gets soiled.

The frequency of hand-washing depends primarily on the nature of the work being done. Outside of work the hands must be washed before every meal, after each visit to the toilet or routine work which soils the hands, before and after sleep.

There are basic, neutral and overfat soaps. Basic soaps contain an admixture of alkali. They soften the skin, but also degrease it, i.e., deprive it of its natural fatty lubrication. Degreased skin becomes dry and irritable. Neutral soap which contains no admixture of free alkali is usually the best for washing the skin. Most types of toilet soap are neutral.

Overfat soap differs from neutral soap in that it contains an admixture of lanolin. It is good for dry and irritable skin which does not always tolerate frequent washing with neutral soap.

People with a dry skin must not wash (especially the face) with cold or hard water. They must wash, particularly the head and face, with rain or snow water, or must add to hard water a little sodium bicarbonate.

The method and frequency of washing the head depend on the condition of the skin on the hairy part of the head. If the skin is healthy, the head is washed once a week with hot water and toilet soap. In cases of dry skin, loss and poor growth of hair the head must be washed once in 10-15 days with hot water and overfat soap. An oily skin requires washing the head once in 4-5 days with neutral and sometimes even with basic soap (green soap, soap liniment).

Baths play a particularly important part in caring for the skin. The temperature of general cleansing baths must be 35-40°C; the baths must last 15-30 minutes.

Proper care of the skin must also make use of the favourable effects of the sun and fresh air. Insolation causes dilatation of the blood vessels, stimulates the receptors of the skin, increases the secretion of the sebaceous and sweat glands and exerts vigorous action on the metabolism in the skin. For example, under the influence of the sun's ultraviolet rays on the skin the antirachitic vitamin D changes from an inactive to an active state. Exposure to the sun tones up the nervous system, improves the general condition and sleep, intensifies metabolism and increases the number of erythrocytes and the amount of hemoglobin. However, insolation produces these favourable effects on the human organism only in cases of reasonable exposure. Excessive insolation

produces contrary results—rapid fatigability, increased excita- bility of the nervous system and disturbed sleep. The first inso- lation in the central zone of the Soviet Union must not exceed 2-5 minutes for each surface (anterior and posterior) of the body. If the person feels good after the insolation, the exposure of each surface of the body to the sun may be increased 5 minutes every day. The maximum total exposure to the sun in the central zone of the Soviet Union may be 2-3 hours a day, the best time being from 9:00 a.m. to 12 noon. It is best to expose the body to the sun after a light breakfast. During the insolation the head must be covered with a hat or a white kerchief, or must be kept in the shade.

A 10-minute rest in the shade after every 30 minutes of inso- lation is recommended.

In the summer-time one may stay in the sun for a longer period (men in shorts, women in corresponding attire) providing one is not lying down but keeps moving. The reason for it is not only that different parts of the body are thus exposed to the sun, but also that the insolation per unit of skin surface is less intense because of the vertical position of the body.

Insolation is contraindicated by severe general diseases, ema- ciation, nervous and endocrine dysfunction, disturbances in car- diac activity, certain forms of pulmonary tuberculosis, and poor tolerance of insolation.

Fresh air also produces very favourable effects on the human organism. Exposure of the naked skin to fresh air (in the shade) tones up the nervous system and improves the blood circulation and secretory function of the skin. Air baths tend to harden the organism and to increase its resistance to colds.

Sports and physical exercise also help to preserve a healthy skin. Physical exercise strengthens and hardens the human orga- nism, tones up the nervous system, intensifies metabolism and improves the skin. After physical exercise it is necessary to wash the sweat and dust off the skin either with a sponge or moist towel and follow it with a mild dry-towel rubdown.

Medical workers must take particular care of their skin. Failure of the medical personnel to observe the rules of hygiene may lead to unpleasant consequences.

CAUSES AND GENERAL SYMPTOMATOLOGY
OF SKIN DISEASES

The skin is most intimately connected with the various organs and systems of the organism, for which reason skin diseases must never be regarded as local affections of the skin, but always as diseases of the whole organism. Dysfunction of the nervous system, diseases of the internal organs, endocrine glands, etc., often produce changes in the skin. The idea that a skin disease is a manifestation of a disease of the whole organism was first expressed by A. Polotebnov who wrote that "a skin disease must never be regarded as a local process" that "it is a general process, a disease of the whole organism".*

Diseases of the skin may in their turn sharply and deeply affect the whole organism. General indisposition and loss of sleep in certain skin diseases which cause itching may serve as an example of such affections.

The causes of skin diseases may be external and internal.

The *external causes* of skin diseases are:

1. *Pathogenic microbes.* Some skin diseases are of infectious origin. Pathogenic microbes gain entrance into the skin directly from the external environment or are carried by the blood from a focus of infection already existing somewhere in the organism.

The infectious, microbial skin diseases include pyodermas, tuberculosis of the skin, leprosy, anthrax, glanders, and viral diseases of the skin.

2. *Vegetable parasites—pathogenic fungi.* Invasion of the skin by pathogenic fungi from the external environment gives rise to such skin diseases as trichophytosis, microsporosis, favus, epidermophytosis, actinomycosis, etc.

3. *Animal parasites.* Some animal parasites, for example, the scab mite, certain protozoans (causative agent of cutaneous leishmaniasis or Borovsky's disease) and hairworms may cause skin diseases by invading the skin. Other animal parasites—lice, fleas, bedbugs, mosquitoes, sandflies, etc., cause skin diseases by their bites.

4. *Mechanical action on the skin*—friction, bruises, pressure, stretching—may cause excoriations, abrasions, hyperemia, edema, hemorrhages and callosities.

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* A. Polotebnov. *Concerning the Theory of Erythemas. Collection of Articles Dermatological Research,* St.Petersburg, 1885, pp. 28-144.

5. *Action of heat and cold on the skin*. Intense heat causes burns of various degrees—from hyperemia to necrosis and even charring of the skin. Prolonged action of moderately intense heat leads to stable dilatation of the capillaries in the skin. Cold produces frost-bite and chills.

6. The following forms of *radiant energy*—sunlight, ultraviolet radiations emitted by a mercury vapour lamp, roentgen rays, radium and radioactive isotopes—may also injure the skin.

7. *Chemical substances* which irritate the skin are acids, alkalis, salts, organic solvents (acetone, etc.), certain dyes, products of oil refining (kerosene, benzine), etc. They also include medicinal substances capable of irritating the skin—mercurials, tar preparations, etc. Some of these substances are absolute irritants and on coming in contact with the skin always cause pathologic changes in it. They include croton oil, strong acids (sulfuric, nitric, hydrochloric, etc.) and alkalis (potassium hydroxide and sodium hydroxide). Other chemical substances are but mild irritants. Their irritating effect depends on individual characteristics of the organism, the condition of the skin at the moment of action of the particular chemical substance and the duration of this action. Such chemical substances, for example, turpentine, certain dyes, various products of coal processing and oil refining, do not always (and not in all people) cause skin diseases.

The resistance of the organism to the causative agents of skin diseases greatly varies in different people. It can be observed, for example, that of several people living under the same conditions some always develop furuncles, others never have them. The resistance of the organism to vegetable and animal parasites also greatly varies. There are people in whom the bites of mosquitoes, sandflies or other insects are always accompanied by a strong skin reaction—redness, edema and prolonged itching.

A. Polotebnov noted the individual reactions of the skin to various stimuli. "Everybody knows", he wrote, "that the skin of different people reacts differently to the selfsame stimulus... This difference is particularly noticeable, for example, in case of bites by parasites (bedbugs, fleas)"*.

Thus the results of the action of external stimuli on the skin depend not only on the strength and duration of the action, but also on the reactivity of the organism. In cases of dysfunction of the central nervous system external causes provoke skin diseases more easily and quickly than they do in cases of a normally functioning nervous system.

M. Petrova demonstrated that it is possible to provoke skin diseases, including furunculosis, in dogs by producing experi-

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* A. Polotebnov. *Concerning the Theory of Erythemas. Collection of Articles Dermatological Research*, St. Petersburg, 1885, pp. 28-144.

mental neurosis in them. On treating these dogs with bromides and other agents for neurosis she observed the furunculosis and other skin diseases to disappear. If experimental neurosis was reproduced in such dogs, the furunculosis recurred. Pyogenic cocci which cause furunculosis are always present on the skin of dogs, but in cases of dysfunction of the central nervous system they are in a position to multiply vigorously and cause furuncles.

The *internal causes* of skin diseases may be:

1. *Metabolic disturbances.* Deposition of urates in the skin in cases of gout, of calcium salts in calcium metabolism disturbances and of cholesterol in lipoid metabolism disorders may serve as examples.

2. *Dysfunction of endocrine glands.* Skin diseases are very often caused by diabetes, thyroid diseases, dysfunction of the gonads and other endocrine disorders. Seborrhea and acne vulgaris not infrequently arise as a result of dysfunction of endocrine glands at the time of sexual maturation.

3. *Diseases of the internal organs.* Skin diseases may develop in persons affected with helminthiasis and diseases of the liver, stomach, intestines, kidneys, lungs and other internal organs. Improvement in the main disease often leads to improvement or disappearance of the skin disease.

4. *Avitaminosis or hypovitaminosis.* Vitamin deficiency may lead to skin disease which develops particularly often in cases of deficiency in vitamin PP (nicotinic acid), vitamin A, vitamin C (ascorbic acid) and vitamins B_1 and B_6.

5. *Consumption of certain foodstuffs and administration per os or parenterally (intravenously, intramuscularly, subcutaneously, etc.) of various drugs.* In these cases the skin diseases are caused by *external* stimuli—foodstuffs, drugs, etc. However, their ability to provoke a skin disease is manifested not as a result of direct external contact with the skin, but only after administration into the organism through the digestive or respiratory tracts, or subcutaneously, intramuscularly or intravenously.

Skin diseases caused by foodstuffs or drugs do not develop in all people. In some people each intake of one sulfonamide pill provokes a skin disease (fixed sulfonamide erythema). At the same time most people harmlessly tolerate large doses of this preparation. In these cases an important part is played by the individual properties of the organism.

Pathologic reflexes are important internal causes of skin diseases. For example, a liver disease or helminthiasis is accompanied by corresponding stimulation of the receptors of the liver or the intestines. The continuous transmission of stimuli from the liver or intestines to the central nervous system produces foci of pathologic excitation which leads to disturbances in the normal activity of the cerebral cortex and the subcortical centres. As a result, the

cerebral cortex sends pathologic impulses to the skin, the impulses disturb the normal activity of the skin and provoke skin diseases.

Some skin diseases are characterised by an *allergic* state. Allergy is an increased sensitivity of the organism to certain substances of external or internal origin. Such substances—*allergens*—may be some foodstuffs (lobsters, strawberries, chicken eggs, etc.), chemical substances (nickel and chromium salts, certain dyes, artificial tars, etc.), medicaments (penicillin, sulfonamides, streptomycin, etc.) and various substances of internal origin. In patients with increased sensitivity to some allergen even a negligible amount of this substance may provoke a skin disease.

Investigations of Soviet scientists have shown that in the development of allergy a certain part may be played by a nervous reflex mechanism.

Unfavourable social and economic conditions are also in large measure responsible for the development of skin diseases.

In countries where the population has poor housing, is malnourished, suffers all sorts of privations, and medical aid is out of reach of the majority of the working people, there is fertile ground for the development and spread of various diseases, including skin diseases. Unhygienic working conditions at industrial enterprises may give rise to occupational skin ˒diseases. Improved living and cultural standards of the population and adequate public health services lead to a sharp drop in the morbidity rate. In the Soviet Union the skin disease incidence has considerably decreased.

GENERAL SYMPTOMS OF SKIN DISEASES

There are subjective and objective symptoms of skin diseases.

The *subjective symptoms* accompanying many skin diseases include itching, pain, burning, sensation of "crawling", numbness, tingling, constriction and tension of the skin. The intensity of the sensations depends on the character of the disease, the extent of pathologic changes in the skin and the state of the nervous system.

The *objective symptoms* of skin diseases are physical manifestations of pathologic changes in the skin. They may have to do with general properties of the skin (colour, turgor, dryness, etc.) or be of the nature of *skin eruptions.*

Every skin eruption consists of various *morphological lesions—primary and secondary.*

Pathologic structures appearing on a healthy skin as effects of harmful internal or external influences are called *primary morphological lesions.* For example, contact with urtica (nettle) causes

the appearance of wheals on the theretofore intact skin. Scald-
ing may result in redness and formation of blisters on the skin. In-
vasion of the skin by tubercle bacilli leads to development of
tubercles. All these lesions—wheals, blisters and tubercles—are
examples of primary pathologic lesions.

Eruptions which are the result of further changes in primary
lesions are called *secondary morphological lesions*. For example,
itching at the site of the wheal resulting from contact with urtica
may force the patient to scratch the wheal, thereby producing an
abrasion which is followed by formation of a crust. The abrasion
and crust are secondary lesions since they have formed as a result
of the successive changes in the primary pathologic lesion—the
wheal. After a burn the blister may open with an erosion forming
at its site. The tubercle may undergo necrosis and develop into
an ulcer. The erosion and ulcer are also secondary pathologic
lesions.

Primary Morphological Lesions

The primary lesions include macules, nodules, wheals, tuber-
cles, vesicles, blisters and pustules.

1. A **macule** is a discoloration of a circumscribed area of the
skin. It is not elevated above the surface of the skin, does not
differ in consistency from the surrounding skin and does not change
the normal pattern of the skin.

The following macules are distinguished: (a) vascular, (b) hem-
orrhagic, and (c) pigmented.

Vascular macules may be of an inflammatory and non-inflam-
matory character. Inflammatory vascular macules appear as a
result of dilatation of superficial blood vessels of the skin. At
first they are pink-red, but later assume a purplish or brownish
hue. Inflammatory vascular macules up to a finger nail in size
are called a *roseola*, larger ones are called an *erythema*. Roseolas
in syphilis, typhoid fever, measles, and erythemas caused by in-
solation are examples of inflammatory vascular macules. Fresh
macules of this type disappear under pressure of a finger or glass
and reappear as soon as pressure is discontinued. Older macules
do not become completely discoloured, but leave a yellowish spot
at their site. This is due partly to the fact that the erythrocytes
leave the blood stream at the site of the macule and the hemo-
globin is gradually transformed into other pigments. Non-inflam-
matory vascular macules arise as the result of a stable dilatation
of blood vessels; they include telangiectases and vascular birth-
marks.

Hemorrhagic macules are characterised by marked extravasa-
tion of blood from the vascular bed into the tissue of the skin.
Their colour gradually changes from red (in the beginning) to
greenish and yellowish. These changes in colour are due to disinte-

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gration of hemoglobin and formation of other pigments from it.

Pigmented macules are changes in the colour of the skin due to an increase or decrease in the amount of pigment on a circumscribed area of the skin. An increase in the amount of pigment produces hyperpigmented macules. They may be congenital (pigmented birthmarks) or acquired (freckles, pigmented macules

Fig. 8. Diagram of the structure of a papule (from A. Zenin and N. Torsuyev)

Fig. 9. Diagram of the structure of a tubercle (from A. Zenin and N. Torsuyev)

of pregnancy). In cases of decreased pigment depigmented macules, also congenital or acquired, are observed. The congenital depigmented macules are white birthmarks, the acquired—vitiligo macules.

2. **Nodule** or **papule**. A papule is one of the primary skin lesions, a circumscribed solid elevation due to thickening of some layer of the skin or accumulation of cellular infiltrate without a cavity (Fig. 8).

Papules may be of an *inflammatory character* if they are formed by accumulation of cells of an inflammatory infiltrate, hyperemia or edema (for example, papules in eczema, psoriasis, syphilis).

Papules of a non-inflammatory character are formed by thickening of some layer of the skin (warts, etc.). They vary in size from that of a pinhead to that of a lentil or pea and even larger. They are most commonly of a more or less dense consistency, and may be flat or conical, round or polygonal.

Papules the size of a lentil or pea are called lenticular papules. Larger papules—the size of a small coin—are known as nummular papules. Still larger lesions formed by a union of several papules into a single continuous focus are referred to as *patches*.

Small papules—the size of a poppy or millet seed—are called miliary papules. They are often pointed. Upon healing the papules disappear without leaving a trace or leaving temporary pigmentation.

3. A **wheal** is also an elevation above the level of the skin without a cavity. Unlike a papule, however, a wheal is formed not by an increase in the number of cells, but as a result of a circumscribed edema. A wheal is usually semispherical and of a soft

consistency; it varies in size from that of a lentil to that of the palm and even larger. Wheals may be red, pink coloured or white, depending on the extent of the edema. A wheal is characterised by rapid development; it appears quickly, is accompanied by intense itching and burning, and usually disappears as quickly without leaving a trace.

4. A **tubercle** is also a cavityless structure elevated above the level of the surrounding skin and consisting of cellular infiltrate. However, the infiltrate of a tubercle is always localised in the derma and on subsequent development undergoes necrosis and is replaced by cicatricial tissue. The size of tubercles varies from that of a millet seed to that of a pea (Fig. 9).

Tubercles vary in consistency. For example, in tertiary syphilis the tubercles are denser than the surrounding skin, but there are also soft tubercles, for example, those in lupus. Necrosis of the infiltrate results in formation of an ulcer at the site of the tubercle; the ulcer heals by forming a scar. If the necrotised tissue is resorbed without forming an ulcer, only cicatricial atrophy of the skin remains at the site of the tubercle.

5. A **node** may be of an inflammatory and non-inflammatory nature. The structure of an inflammatory node greatly resembles that of a tubercle, but the infiltrate of the node is situated deeper, in the subcutaneous adipose layer. The infiltrate very often also affects the derma. The size of a node varies from that of a bean to that of a chicken egg and even larger (Fig. 10).

Fig. 10. Diagram of the structure of a node (from A. Zenin and N. Torsuyev)

Fig. 11. Diagram of the structure of a vesicle (from A. Zenin and N. Torsuyev)

On healing of a node the infiltrate in most cases undergoes necrosis which leads to development of an ulcer followed by formation of scar tissue. A syphilitic gumma, scrofuloderma and furuncles are examples of such nodes. In other cases, for example, in erythema nodosum, the cellular infiltrate does not disintegrate but is gradually resorbed and no scar is formed.

Fibroma and lipoma are examples of non-inflammatory nodes.

6. A **vesicle** is a structure with a cavity containing a transparent or slightly turbid serous fluid. It is elevated above the level

of the surrounding skin and varies in size from that of a pinhead to that of a pea. A vesicle may be covered either by the entire thickness of the epidermis or only by its upper layers, sometimes merely by the stratum corneum (Fig. 11). Subsequently vesicles either burst, forming an erosion, or their content gradually dries and a crust is formed.

On the palms and the soles of the feet vesicles often fail to raise the overlying layers of the epidermis because of their considerable thickness and are merely seen through them as sago grains. Groups of vesicles developing on a small area of the skin are called *herpes* or *herpetic vesicles*.

7. A **blister** resembles a vesicle in structure, but is larger. Blisters vary in size from that of a hazelnut to that of a chicken egg and even larger. Both blisters and vesicles may be *unilocular* or *multilocular*. Puncture of the cover of a unilocular blister results in emptying of the entire cavity. In multilocular vesicles the cavity is divided into "chambers" by partitions, and a puncture results in but partial draining. Blisters and vesicles heal without forming scars.

8. A **pustule** is a locular structure, but unlike vesicles it contains purulent matter. If a pustule is in the epidermis, it is considered superficial, but if it penetrates into the derma, it is regarded as deep.

The difference between superficial and deep pustules tells on their outcome; after healing a superficial pustule leaves only a temporary pigmentation, whereas a deep pustule always leaves a scar.

Secondary Morphological Lesions

The secondary morphological lesions are: scales, crusts, erosions, excoriations, fissures, ulcers, scars, atrophy, secondary pigmented macules, lichenisation and vegetations.

1. **Scale**. The horny lamina located in the upper part of the stratum corneum gradually become disconnected from the deeper lamina and are continuously cast off. Physiologic desquamation is scarcely visible to the eye, but can be detected by stroking the skin with the hand wearing a dark knitted glove. Disturbances in the process of cornification sharply increase the desquamation of horny laminas from the surface of the epidermis and they are cast off in layers as scales.

A *scale* is a particle of the stratum corneum cast off from the epidermis. It often contains dust, dirt and secretion of the sweat and sebaceous glands. There are two types of desquamation—*branny* and *laminar*.

Branny scales are small and resemble flour or bran. Laminar scales are larger and look like laminas. Scales are very often ob-

served in resolution and in resorption of primary morphological lesions.

2. A **crust** is a dried exudate. Crusts are formed by the drying contents of vesicles, blisters and pustules, the disintegrated matter of tubercles and nodes, and the discharge from erosions, ulcers and excoriations. There are serous, purulent and sanguineous crusts. Admixtures of dust, dirt and medicaments may change the colour and appearance of the crusts. When a crust is cast off it leaves, in some cases, a temporary pigmentation and, in others, a scar or cicatricial atrophy of the skin.

3. An **erosion** is a superficial (within the epidermis) defect of the skin arising at the site of a vesicle, blister or pustule after the bursting or removal of their cover. Sometimes erosions are formed at the site of papules. The shape and size of an erosion correspond to the shape and size of the lesion at whose site it has formed. An erosion has a smooth and sometimes bleeding floor. It heals by epithelisation without forming a scar.

4. An **excoriation** resembles an erosion but is due to mechanical disruption of the continuity of the epidermis as a result of injury or scratching. The discharge from an excoriation is often sanguineous because the defect in the epidermis may reach the papillary layer of the derma. Excoriations become covered with serosanguineous or sanguineous crusts.

5. A **fissure** may be the result of mechanical disruption of the continuity of the skin through its excessive stretching. Usually fissures arise on the skin that has lost its normal elasticity in inflammatory processes, in cases of xeroderma and in old age. Most commonly fissures are linear. They are always superficial when occurring only within the epidermis. Deep fissures project into the derma and often bleed.

6. An **ulcer** is a defect in the skin projecting into the derma and forming as a result of tissue necrosis. Ulcers often spread to the subcutaneous tissue and sometimes even to deeper tissues—muscles, periosteum and bones. Ulcers usually develop from tubercles and nodes, but under special conditions may also develop from other morphological lesions. Ulcers vary in shape, size and depth and always heal by forming a scar. The floor of an ulcer may be covered with granulations, purulent or serous matter, or a necrotic film.

7. A **scar**—a coarse, fibrous, connective tissue structure—arises at the site of a destroyed connective tissue structure of the derma, most commonly at the site of a tubercle, node or ulcer. In cases of deep tissue destruction the scars are drawn in and adhere to the underlying tissues.

Scar tissue is deficient in blood vessels. At first scars are reddish, reddish-blue, and then become almost white. If a scar is elevated above the level of the surrounding skin, it is called

in morphology, if it is below the level of the surrounding skin, it is called atrophic. The surface of a scar is smooth and it has no skin fields, furrows, sebaceous or hair follicles, or sweat glands.

8. **Cicatricial atrophy of the skin** is characterised by thinning, dryness and fine cutaneous folds. Atrophy develops at the site of infiltrates caused by destruction of the normal structure of the derma. If no ulcer is formed after destruction of the connective tissue basis of the derma and its replacement by coarse connective tissue, the result is not a scar, but cicatricial atrophy. Cicatricial atrophy may develop at the site of tubercles and sometimes nodes.

The areas of cicatricial atrophy have no skin furrows or fields, no hair and no sebaceous or sweat glands. Atrophic areas easily form very fine folds and resemble creased cigarette paper. Subcutaneous veins can often be seen through thinned atrophic skin.

9. **Secondary pigmented macules,** or pigmentation, develop after disappearance of various primary and secondary morphological lesions. They remain at the site of tubercles, macules, nodules, vesicles, erosions and other lesions. Such, for example, are pigmented macules at the site of a former morbilliform eruption, syphilitic roseolas and papules, etc.

10. **Lichenisation.** In cases of lichenisation the skin thickens, becomes more compact to touch and darker because of secondary pigmentation. The skin pattern becomes coarse, the furrows deepen and the skin fields are more sharply defined. At the sites of lichenisation the skin is covered with scales. Lichenisation occurs mainly in chronic skin diseases.

11. **Vegetations** are papillary growths which sometimes occur on papules, floors of ulcers and erosions, on nodes and tubercles.

Examination of the morphological lesions is very important for establishing a correct diagnosis, evaluating the course of the skin disease and making a prognosis. Neither timely diagnosis of skin diseases nor their correct treatment is possible without knowledge of the morphological lesions.

Examination of a patient affected with a skin disease must reveal:

1. The primary and secondary morphological lesions of the eruption.

Monomorphic and polymorphic eruptions are distinguished. A monomorphic eruption consists of morphological lesions of only one type. Roseolas in typhoid fever, ordinary warts, papules in psoriasis and in lichen ruber planus may serve as examples of a monomorphic eruption.

A polymorphic eruption is characterised by the presence of several types of morphological lesions. A true and false polymorphism of eruption are distinguished. In true polymorphism there are simultaneously several types of primary lesions. For example, during the secondary stage of syphilis the eruption may consist

of roseolas, papules and pustules. In false polymorphism one type of lesion is observed to change to another. For example, in acute eczema the process begins with formation of macules which are soon transformed into papules and then vesicles. The vesicles open and form erosions which may become covered over with crusts. Consequently, in acute eczema all the various morphological lesions are but different stages of development of one lesion.

2. The shape, size, colour and consistency of the lesions present.

3. The number and distribution of the lesions over the skin; whether or not the eruption is limited to one or several areas or is spread over a large surface of the skin. In cases of diffuse lesions it is necessary to see whether the eruption is symmetrical or asymmetrical.

4. The mutual arrangement of the morphological lesions; whether they occur in groups or are scattered over the skin, and whether the various lesions have coalesced. Coalescence of the lesions is a sign of their peripheral growth.

5. The course of the pathologic process, for which purpose it is necessary to question the patient as to the appearance of the primary lesions of the eruption and the changes they subsequently undergo.

GENERAL PRINCIPLES OF TREATING SKIN DISEASES

Skin diseases are, as a rule, diseases of the entire organism. Even when a skin disease is due to the effects of external causes it becomes a disease of the entire organism and is not only a local process. Scabies may serve as an example. Having lodged itself in the skin the scab mite which nestles in the outermost layer of the epidermis causes appreciable changes in the general condition of the organism—intense itching, scratching and insomnia. It stands to reason that the disturbances in the functions of the nervous system and the internal organs greatly differ in the different skin diseases.

In treating patients affected with skin diseases it is always necessary to take into account the disturbances in the general condition of the organism. A correct approach to the treatment of a skin patient is based on a careful examination of the patient's general condition with due regard for all conditions of his external environment, i.e., his working and living conditions. This makes it possible to ascertain the factors which provoke and maintain the skin disease in the given patient.

Elimination of the causes responsible for skin diseases is the most important part of the treatment. For example. in some cases of urticaria the cause of the disease may be the presence of helminths in the intestines. If this is discovered and the helminth are expelled, the urticaria often disappears without any additional treatment.

It is no less important to eliminate everything that predisposes the organism to the skin disease and maintains this disease. In some cases chronic gastritis, helminthiasis, a tendency to constipation or chronic colitis do not provoke a skin disease, but predispose the organism to it by weakening the organism and altering its reactivity. If the skin of such a patient comes in contact with some external stimulus, the organism may react by developing a skin disease.

A "predisposition" may sometimes explain why, while working under the same conditions, one worker develops an occupational dermatitis or eczema on contact with nickel salts, turpentine or some other substances and the other workers do not. In such a case elimination of the predisposing factors, for example, gastrointestinal diseases, etc., is no less important than elimination of the external cause, i.e., the action of the chemical substance on the skin.

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All therapeutic influences exerted in the treatment of patients affected with skin diseases may be divided into *general* and *local* (*topical*).

As a rule, skin patients must be given *overall* treatment. An appropriate regimen as well as general and local treatment are prescribed with due regard for the causes and nature of the skin disease, the patient's general condition and the conditions of his life and work.

General Treatment of Skin Diseases

General treatment of skin patients is treatment of the entire organism. It is necessary in almost all cases of skin disease. Good results can be produced by local treatment in comparatively few cases of skin diseases. Various methods and agents known to modern medicine are widely used in the general treatment of skin diseases. They include:

1. *Influences exerted on the central nervous system.* These influences may be produced by the use of novocain, bromides, neuroplegic and ganglion blocking agents, and other methods. Novocain may be administered: (a) intravenously in a daily dose of 2-10 ml of a 0.25 per cent solution*, a total of 20-30 injections per course of treatment; (b) in the form of a circular block: 50-100 ml of a 0.25 per cent solution is administered intracutaneously and then subcutaneously around the entire extremity (above the focus of affection); (c) per os in a dose of 1-3 tablespoonfuls of a 0.25-0.5 per cent solution one hour before meals, 3 times per day for 4-6 weeks.

Bromides are administered per os in a dose of 1 tablespoonful of a 0.25-2 per cent solution 2-3 times per day before meals. Sodium bromide is also administered intravenously in a daily dose of 5-10 ml of a 10 per cent solution.

Treatment with ganglion blocking and neuroplegic agents is administered in hospitals for skin and venereal diseases.

Skillful psychotherapy is very important in the treatment of skin diseases. The medical workers must encourage and reassure their patients and convince them that they will recover.

2. *Desensitising therapy.* The nonspecific desensitising agents which reduce the organism's sensitivity to a number of stimuli include calcium chloride, sodium hyposulfite and certain other preparations.

Calcium chloride is administered intravenously as a 10 per cent solution in a dose of 5-10 ml daily or every other day (a total of 10-15 injections per course of treatment), or per os in a dose of 1 tablespoonful of a 10 per cent solution 3 times per day.

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*Here and further on the doses are indicated for adults. For children and old people the doses vary with age.

Sodium hyposulfite is administered intravenously in a daily dose of 10 ml of a 10-30 per cent solution (a total of 15-20 injections per course of treatment), or per os either in a dose of 1 tablespoonful of a 10 per cent solution or as a powder of 0.5-1 g 3 times per day before meals.

3. *Antihistaminic preparations.* The histamine and histamine-like substances in the skin increase in a number of skin diseases. This causes itching and is responsible for the dilatation of the blood vessels, development of edema and formation of vesicles. In such cases antihistaminic preparations weaken the organism's reaction to histamine and similar substances, diminish the itching and abate the inflammatory phenomena. Antihistaminic substances include dimedrol (diphenhydramine), diazoline (5-benzyl-1,2,3,4-tetrahydro-2-methyl-SH-pyride-indole), diprazine (N-/2-dimethylaminopropyl/phenothiazine hydrochloride), etc.

Dimedrol is administered per os in a dose of 0.03-0.05 g 3 times per day or intramuscularly in a dose of 1-5 ml of a 1 per cent solution. Diazoline is given per os in a dose of 0.1-0.2 g 1-2 times per day. Diprazine, promethazine and phenergan are also given per os in a dose of 0.025-0.05 g 2-3 times per day.

4. *Immunotherapy.* Immunobiological methods of treating skin diseases are aimed at strengthening the processes of immunity in the patient's organism. Immunotherapy is used in skin diseases with an established or supposed infectious etiology. Immunotherapy may be specific, when bacteriological preparations are used, for example, staphylococcus toxoid or vaccine in the treatment of pyodermas, etc.

Nonspecific immunotherapy includes autohemotherapy, injections of milk and laky (hemolysed) blood. These methods are based on the moderately stimulating effects of the patient's own or foreign protein administered subcutaneously or intramuscularly, which enhances the organism's resistance to infection.

Autohemotherapy is administered once in 3 days. Usually 5 ml of blood is administered the first time, 8 ml the second time, and then 10 ml each time until a total of 4-8 injections has been made.

To inject laky blood, 5 ml of sterilised distilled water is aspirated into a 10-ml syringe, the needle is inserted into the patient's vein and 5 ml of his blood is aspirated into the same syringe. The syringe and needle are then removed from the vein and, by a light shaking, the blood and water are mixed. Within 1-2 minutes the mixture becomes transparent and somewhat laky owing to hemolysis of the red blood corpuscles. After this the mixture is administered to the patient intramuscularly.

In many cases injections of laky blood produce better results than does autohemotherapy, but they are more painful. Laky

blood is administered once in 3-4 days, 4-8 injections per course of treatment.

Lactotherapy. Freshly boiled milk is administered intramuscularly once in 3-5 days, a total of 4-8 times; the initial dose of 3 ml is gradually increased to 7-10 ml. Lactotherapy requires caution because of the strong general reaction (weakness, fever) it may evoke. Sometimes phenomena of anaphylaxis—redness and edema of the mucosa, and cardiovascular disturbances—are observed several minutes after the injection.

A subcutaneous administration of 0.5-1 ml of a 1:1,000 adrenalin solution quickly eliminates all these phenomena, so that further continuation of lactotherapy is not recommended.

5. *Stimulating therapy*. This is the designation given to the methods of treatment aimed at strengthening the defensive powers of the organism, increasing its resistance and enhancing the tone of the nervous system. They include the method of blood and plasma transfusions and gamma globulin injections.

In the treatment of skin patients 50-200 ml of blood of the same group is transfused once in 6-7 days, a total of 3-6 times.

6. *Antibiotics.* One of the antibiotics often used in the treatment of skin diseases is penicillin. It is particularly frequently prescribed for the treatment of pyodermas. Penicillin also possesses the ability to diminish the phenomena of intoxication and improve the patient's general condition in certain skin diseases of noninfectious or unclear etiology as, for example, in arsphenamine erythroderma and scleroderma. It is administered intramuscularly in a dose of 50,000 u every 3 hours or in a dose of 100,000-150,000 u in 2-3 ml of a 1 per cent novocain solution 4 times per day, or in the form of ecmonovocillin in a dose of 600,000 u once a day.

Streptomycin and dihydrostreptomycin are prescribed mainly for the treatment of patients with tuberculosis of the skin.

Synthomycin (chloramphenicol) and levomycetin or chloromycetin (the two latter preparations are also varieties of chloramphenicol) are administered per os in a dose of 0.5 g 3-4 times per day in the treatment of certain forms of pyoderma.

Biomycin (chlortetracycline), tetracycline and terramycin (oxytetracycline) find application in pyodermas and exudative erythema. They are given per os in a dose of 0.2 g 4-5 times per day.

Synthomycin and, especially, biomycin often cause complications—itching, toxicodermas, and yeast lesions in the mucous membranes and the skin. To prevent these side effects, it is necessary, as soon as administration of these antibiotics begins, to prescribe thiamine, riboflavin, nicotinic and ascorbic acids, and in cases of prolonged administration of the above antibiotics, especially to children, elderly people and debilitated patients, also

nistatine (an antiyeast antibiotic) in a dose of 500,000 u 3 times per day. Administration of the aforesaid antibiotics is suspended upon appearance of the first signs of side effects.

7. *Vitamins* should be administered to all patients exhibiting signs of avitaminosis or hypovitaminosis. Vitamins are also prescribed in the absence of signs of vitamin deficiency because some vitamins produce good effects on certain skin diseases. For example, nicotinic acid favourably affects patients suffering from lupus erythematosus, acne rosacea and parapsoriasis, although usually no deficiency in this acid in the patients is observed. It also possesses the ability to reduce the sensation of itching.

Nicotinic acid is administered intravenously or intramuscularly in a daily dose of 2-10 ml of a 1 per cent solution, a total of 15-20 times. Administration of nicotinic acid evokes in the patient a vascular reaction; immediately after the injection the skin, especially on the face and neck, becomes red and sometimes somewhat edematous, the patient feeling hot. These phenomena usually pass off within a few minutes, but the patient must be warned about them.

Nicotinic acid is also given per os in a dose of 0.05-0.1 g 3 times per day after meals for 3-4 weeks.

Vitamin B_1 or thiamine produces favourable effects in cases of dysfunction of the central and peripheral nervous system, and in skin diseases accompanied by itching or pain.

Thiamine is most commonly prescribed intramuscularly or intravenously in a daily dose of 50-60 mg (1 ml of a 5-6 per cent solution), a total of 20-30 injections. It is often also administered per os in a dose of 0.02-0.03 g 3 times per day, usually for 3-4 weeks. Vitamin B_2 or riboflavin is for the most part given per os in a dose of 5-10 mg 3 times per day for about 3 weeks.

Vitamin B_{12} is used in the treatment of lupus erythematosus, certain forms of eczema and skin diseases accompanied by intense itching and phenomena of anemia.

Vitamin A is effective in xeroderma, excessive cornification of the sebaceous and hair follicles and in certain forms of pyoderma. The dose is 10-20 drops of a vitamin A concentrate after meals 3 times a day for 2-3 months.

Vitamin D_2 is prescribed mainly for patients with tuberculosis of the skin.

Ascorbic acid is administered intravenously in a daily dose of 10 ml of a 5 per cent solution (a total of 10-20-30 injections) or intramuscularly in a daiy dose of 2-10 ml of a 5 per cent solution for the same length of time.

Per os ascorbic acid is best to be given in pills of 0.1 g 3 times per day before meals. Ascorbic acid produces the best effects when administered together with vitamin P (0.075 g twice a day) or its preparations—citrin and rutin.

8. *Sulfonamides* are indicated in the treatment of pyodermas and herpetiform dermatitis. The preparations most commonly prescribed are norsulfazole (sulfathiazole), sulfodimesine (2-/para-aminobenzol sulfamido-/4,6-dimethyl piperidine) and ethazole (2-para-aminobenzol-sulfamido/-2-ethyl-3,4-thiodiazole) in a dose of 0.5-1 g 3-4 times a day for 5-7 days, sometimes longer.

9. *Corticosteroids*. Prednisone, prednisolone and triamsinolone are corticosteroids that can be effectively used in certain skin diseases running a severe course and constituting a threat to the patient's life (acute lupus erythematosus, pemphigus), as well as disseminated skin diseases with a clearly marked allergic state and stubborn course (some forms of eczema, psoriasis, etc.). In such cases large doses—40-80 mg of prednisone or prednisolone a day—are usually prescribed in the beginning of the treatment. As the patient's condition improves, the doses are gradually reduced (by 5-10 mg of prednisolone once in 4-7 days). In severe skin diseases (acute lupus erythematosus and pemphigus) the dose is reduced more slowly, a small "supportive" dose (5-15 mg of prednisolone a day) being administered for a long time.

The doses of triamsinolone are smaller because this preparation possesses more vigorous action than do prednisone and prednisolone. The treatment begins with 18-24 mg of triamsinolone a day and the "supportive" dose is about 4 mg a day. Dexametasone is administered in still smaller doses—3-9 mg a day in the beginning of the treatment and about 1.5 mg as the "supportive" dose.

Treatment with large and medium doses of corticosteroids may be accompanied by dangerous complications—adiposis, disturbances in water and mineral metabolism with retention of sodium and water in the tissues, elevation of blood pressure, development of gastric or duodenal ulcer and even its perforation, rarefaction of bony substance, bone fractures, etc. Treatment with corticosteroids must therefore be administered only on prescription and under supervision of a physician, and, best of all, in a hospital. In treatment with large and medium doses of corticosteroids the patient must be prescribed a salt-free diet; the food should be consumed in small portions 5-6 times a day during the same hours; coarse and spicy foodstuffs are prohibited; the food is grated; common salt is excluded and is replaced by calcium chloride (KCl)—about 3 g per day.

10. *Arsenicals*. The favourable effects of arsenic are due to its strengthening and tonic influences on the organism.

Arsenic helps to improve the processes of metabolism and hematopoiesis, produces favourable effects in skin diseases in which the processes of cornification are disturbed, for example, in psoriasis, lichen ruber planus, verruca plana, and in skin diseases combined with anemia and emaciation. Arsenic is administered in the form of subcutaneous injections of a 1 per cent sodium ar-

.....dc .oluticn in a daily dose of 0.25-1 ml, a total of 30-40 injections. Per os arsenic is administered in the form of Asiatic pills (3-5 pills 3 times per day for 3-4 weeks) and Fowler's solution.

Oxygenotherapy. Subcutaneous insufflation of oxygen is used in the treatment of skin and venereal diseases; the dose is 200-500 cc of oxygen insufflated into the interscapular region once in 3 days, 6-15 insufflations by means of two interconnected Bobrov's apparatus and oxygen tent.

Oxygenotherapy improves the patient's general condition and sleep and exerts favourable action on patients affected with keloids and seroresistant syphilis.

Therapeutic feeding. Digestive disturbances play an important part in the development of a number of skin diseases. Proper feeding helps to restore normal digestion. In allergic skin diseases a "desensitising diet" with limited amounts of carbohydrates is helpful. Consumption of carbohydrates is also limited in stubborn furunculosis, hidradenitis and folliculitides. Patients affected with urticaria, pruritus, disseminated acute eczema and neurodermatitis are allowed but limited consumption of salty and spicy dishes, canned foods, eggs and sweets, and are given a dairy and vegetable diet with lots of fruit and greens. Consumption of salt is also limited in cases of tuberculosis of the skin.

It is particularly important to see to it that the patients have regular bowel movements, for which purpose skin patients suffering from constipation are prescribed sour milk, black bread, raw and cooked vegetables.

In skin diseases *treatment at health resorts* is one of the effective methods of therapy, especially if the diseases run a stubborn course and resist other methods of treatment. Health-resort treatment may prove not only useless, but even harmful if the existing contraindications are not taken into consideration.

Health resorts with hydrogen-sulfide springs are particularly widely used in the treatment of skin patients. Good effects in the treatment of skin patients are observed at health resorts with sulfide-carbonate and radon springs and at seashore health resorts.

A favourable general regimen is one of the most important factors of successful treatment of skin diseases. It is necessary to arrange a proper alternation of work and rest. It is very important that the patient should spend part of his leisure—at least 1-2 hours a day—outdoors.

If the patient's general condition does not contraindicate physical exercise, regular indulgence in sports is recommended. Sports strengthen and harden the organism, tone up the nervous system and improve metabolism. Skin patients will greatly benefit by systematic morning exercise followed by a sponging or moist rubdown.

Elimination of unfavourable influences on the patient's mental state is no less important. Prevention of distressing experiences associated with family or business trouble contributes to rapid recovery.

Local Treatment of Skin Diseases

Local or external treatment of skin diseases is very important. It is aimed at expediting the healing of pathologic foci and at eliminating or weakening the unpleasant sensations of itching, burning, tension and contraction of the skin.

The very concept of "local treatment" is conditional. Any locally used agent in some measure affects the entire organism as a whole, i.e., exerts *general* action. Applied externally the medicinal substances act upon the receptors of the given skin area and through them on the central nervous system. Moreover, some external agents are absorbed by the skin and enter the bloodstream.

In skin diseases the agents for external treatment are chosen in accordance with the character of the pathologic process. The more acute the inflammatory process, the more delicate and cautious must the external treatment be. In subacute and chronic inflammatory processes phenomena of edema and exudation are replaced by phenomena of infiltration of the skin. In these cases external treatment must exert deeper and more vigorous action.

Preliminary debridement of the focus of affection. The external treatment of skin patients must begin with preliminary debridement of the affected area. Forceps, scissors and cotton tampons are used to remove from the focus of affection scales, crusts, remnants of vesicles, blisters, pustules and formerly applied medicinal substances. The scales and crusts found on the focus are carefully removed with a forceps. The remnants of vesicles, blisters and pustules, and the closely adhering scales are cut off with curved scissors. The closely adhering crusts and the remnants of formerly applied ointments and pastes are softened by applying a heavy layer of oil and 10-15 minutes later are carefully removed with a forceps and cotton tampon soaked in the same oil. Various oils are used for this purpose—vegetable oils (peach, sunflower-seed, linseed), animal oils (fish liver oil) and mineral oils (vaseline oil). In cases of erosions and ulcers their surfaces are covered with a 3 per cent hydrogen peroxide solution for the purpose of removing the pus, necrotic film, etc. Following this the resultant foam and discharge from the erosions and ulcers are removed by means of moist cotton or gauze tampons. The hair on the affected area is clipped with scissors. The skin surrounding the focus of affection is usually rubbed down with alcohol or oil.

The medicinal substances for external treatment of skin diseases may be used in various *medicinal forms*. The medicinal form is the physical state in which the medicinal substance is used.

The following medicinal forms are most commonly used in the treatment of skin diseases: powders, lotions, coatings, moist dressings, water suspensions, pastes, oils, ointments, compresses and plasters. Here the medicinal forms are arranged according to the depth of their action; powders produce the most superficial effects, compresses and plasters—the deepest (Fig. 12).

Name of the medicinal form	Constituents of the medicinal form	Indications for use
Powders	▨	Acute inflammatory processes in the erythe-matous stage
Lotions 1. Cooling 2.Moist-desiccant 3.Dermatological compress	⊜	Acute inflammatory processes in the stage of vesiculation, exudation and edema
Water suspensions	▨ + ⊜ (20-50%)(50-80%)	Acute and subacute inflammatory processes without marked exudation
Pastes	▨ + ⬭ 1:1	Acute and subacute inflammatory processes without vesicles, exudation and crusts
Oil suspensions	▨ + ▱ (20-40%)(60-80%)	Acute and subacute inflammatory processes without vesicles and exudation
Ointments	⬭ + ▦	Chronic and subacute inflammatory processes

▨ Powdered substances   ⊜ Water solutions

▱ Thin fatty bases   ⬭ Fatty bases

▦ Various medicinal substances

Fig. 12. Main medicinal forms for external treatment
of skin diseases

1. *Powders* are applied to the affected area in a thin, even layer. Indifferent powders exert no chemical action on the skin and are used only because of their physical properties—their great absorbing ability. They are prescribed in acute inflammatory processes with phenomena of hyperemia, moderate edema and tension of the tissues. Fine powders very well absorb the exudate oozing out of the inflamed skin, the sebum and sweat. The moisture absorbed by a powder evaporates. Indifferent powders dry and cool the skin and exert an anti-inflammatory action. These powders include mineral powders (zinc oxide, talcum and kaolin) and vegetable powders (wheat, rice and potato starch).

The active powders include disinfecting powders—xeroform, dermatol (bismuth subgallate), streptocide and other sulfonamides used in the treatment of ulcerated and eroded surfaces.

2. *Lotions* are made of water solutions of medicinal substances. A piece of gauze or clean linen folded in 2 or 3 is soaked in the medicinal solution, slightly wrung out and applied to the affected area. As the application dries, the gauze is soaked and wrung out again. The medicinal solution must be of room temperature or somewhat colder. The indifferent or weakly astringent and weakly disinfecting lotions include 0.25-0.5 per cent silver nitrate solutions, 1 per cent aluminium acetate solution (Burow's solution), lead water, and 1-2 per cent resorcin (resorcinol) solution. Lotions are used in cases of acute inflammation—hyperemia, edema and exudation.

The effects of lotions consist in cooling the inflamed area, constriction of dilated blood vessels and increased evaporation of exudate. The astringent and disinfecting influence of a medicinal solution increases the anti-inflammatory action of the lotion. In cases of exudation and pyogenic infection lotions with stronger disinfecting action are used, namely, rivanol (2-ethoxy-6,9-diaminoacridine lactate) solution (1:1,000), methylene blue (1:1,000) solution and furacilin (5-nitro-2-furaldehyde semicarbazone) solution (1:5,000).

3. *Moist-desiccant dressings* are a variety of a cooling lotion. For this purpose 8-12 gauze napkins or pieces of linen are soaked in one of the aforementioned solutions and are applied to the affected area. If necessary, they are covered with a dressing of several turns of bandage. Moist-desiccant dressings are changed as they get dry, within 30-60 minutes.

A "dermatological compress" is a variety of a moist-desiccant dressing. The compress consists of 10-15 layers of gauze or linen soaked in a medicinal solution. It is applied to the affected area, is covered with a piece of wax-paper or compress oilskin and is fastened with a bandage (without cotton). The compress is changed 3-4 times a day. A "dermatological compress" exerts deeper action than do the usual lotion and moist-desiccant dressing.

4. *Coatings*. Water as well as alcohol solutions of various medicinal substances are used for coating. Silver nitrate in the form of a 2-10 per cent water solution is often prescribed for coating erosions in the oral cavity, areas of stubborn pinpoint exudation (chronic eczema) and ulcers with excessively luxuriant granulations.

Alcohol solutions are used for disinfecting the skin in pyodermas (alcohol-camphor solution, 2 per cent alcohol salicylate, etc.). To abate the itching in pruritic skin diseases (urticaria, prurigo, etc.), 1-1.5 per cent alcohol solutions of carbolic acid, menthol, and thymol are used. Solutions (1-2 per cent) of aniline

dyes (methyl violet, brilliant green, etc.) are also prepared with alcohol.

5. *Water suspensions* are mixtures of about equal amounts of liquid and powdered substances. The latter include zinc oxide, talcum, kaolin and starch. Glycerin and water form the liquid part of water suspensions. When allowed to stand suspensions always divide into two layers—the lower, of powdered substances, and the upper, the liquid. Before use the suspensions are well shaken until a uniform suspension, resembling sour cream, is produced. This suspension is applied with a piece of cotton in an even layer to the foci of affection. The liquid part of the suspension evaporates thereby reducing the heat, cooling the skin and exerting anti-inflammatory action on it. After evaporation of the liquid a thin layer of powdered substances remains on the skin. These substances absorb the inflammatory exudate, sebum and sweat, and reduce the sensations of tension, burning and itching in the affected area of the skin. A suspension thus combines, as it were, the effects of a powder and a lotion. Suspensions are used in acute and subacute inflammatory processes involving hyperemia and moderate edema. Exudation contraindicates prescription of suspensions because exudate may accumulate under the layer of the dried suspensions, disintegrate and irritate the skin.

The water suspensions exert a somewhat deeper action than do powders and lotions, but their action is superficial just the same and in subacute and chronic processes with an infiltrate in the derma and the subcutaneous tissue they are of little effect. If the cooling effect of a water suspension has to be increased, alcohol is added to it.

If necessary, medicinal substances—ichthyol (1-5 per cent), anesthesin (ethyl aminobenzoate—2-5 per cent), sulfur (1-5 per cent), etc.—are added to the water suspensions.

6. A *paste* is a mixture of about equal amounts of powders and a fatty base.

Pastes have the consistency of dough and adhere to the skin without a dressing. They exert a somewhat deeper action than do water suspensions. Since they contain a lot of powder they very well absorb exudate and do not hinder evaporation from the surface of the skin.

Pastes are prescribed for acute and subacute inflammatory processes without marked edema and exudation. They may not be used on the hairy parts of the body because they glue the hair and may irritate the skin by the exudate accumulating under the glued hair. One of the most commonly used indifferent pastes is zinc paste. With an addition of 2 per cent salicylic acid it is called Lassar's paste. In other cases ichthyol (1-3 per cent), sulfur (1-5 per cent), tar (1-10 per cent) or naphthalan oil (5-50 per cent) are added.

Pastes are applied with a spatula in a thin layer directly to the skin or to a piece of cloth; the latter cases require a dressing. The layer of paste is renewed once or twice a day without removing the remains of the preceding layer. The entire layer of paste is removed once in 3 days. To remove the paste, the area covered with paste is first coated with a heavy layer of vegetable oil and 10-15 minutes later is carefully wiped with cotton tampons soaked in the same oil.

7. An *oil suspension* is a mixture of one or several powders (zinc oxide, talcum, kaolin, etc.) and a liquid fatty base. The powders constitute 20-40 per cent of the entire mass. The liquid fatty bases are most commonly vegetable oils—peach, apricot, sunflower-seed, and less frequently vaseline oil. The most widely used oil suspension is the so-called "zinc oil"—a suspension of zinc oxide in oil.

Before application the suspension is vigorously shaken and then applied with a piece of cotton to the affected areas. Oil suspensions absorb exudate, soften the skin and relieve the sensation of tension and contraction. They are indicated in acute and subacute inflammatory processes in which there is no exudation. Their action is somewhat deeper than that of pastes.

Various medicinal substances—ichthyol (1-5 per cent), sulfur (1-10 per cent), tar (1-2 per cent), etc.—are often added to oil suspensions.

8. An *ointment* is one of the most commonly used medicinal forms of external treatment of patients affected with skin diseases. It consists of a fatty or nonfatty (synthetic) base and one or several medicinal substances evenly mixed with the base. The ointment is applied to a piece of cloth or gauze in an even layer 2-3 mm thick. The dressing is fastened with a bandage. Sometimes the ointment is applied directly to the skin without a dressing, especially to exposed parts of the body, such as the face. In some cases the ointment is rubbed into the skin, for example, in the treatment of scabies.

Ointments penetrate into the ostiums of sebaceous follicles and sweat glands, and deep into the stratum corneum. The even layer of ointment forms an airtight coat on the surface of the skin thereby sharply reducing evaporation of water from it. Water vapours are retained under the layer of ointment and heat the skin. This leads to loosening of the epidermis and the loosened epidermis becomes more permeable to medicinal substances contained in the ointment. Hence the deeper action of ointments than of powders, lotions, suspensions and pastes.

The deep action of ointments makes possible their wide utilisation in the treatment of patients having chronic inflammatory processes with a cellular infiltrate coming to the foreground in the derma or subcutaneous adipose tissue.

The action of an ointment largely depends on the medicinal substances contained in the fatty base. Ointments may be indifferent if indifferent substances are added to the fatty base. For example, the well-known zinc ointment contains 10 per cent zinc oxide in lard or vaseline. Ointments containing corticosteroids (1-2.5 per cent hydrocortisone, 0.5 per cent prednisolone) possess anti-inflammatory and antipruritic activity.

Disinfecting ointments include ammoniated mercury ointment (2-5 per cent), and ointments containing yellow mercuric oxide (1-3 per cent), penicillin (1,000-5,000 u per 1 g of base), xeroform (5-10 per cent), dermatol (5-10 per cent), synthomycin (chloramphenicol—1-3 per cent), chlortetracycline (3 per cent), etc.

Ointments containing salicylic acid (3-10 per cent), resorcinol (3-5 per cent), sulfur (10-30 per cent) and certain other substances are exfoliative ointments.

Ointments containing substances conducive to resorption of infiltrate are used in chronic skin diseases with infiltrate in the deep layers of the skin; such substances are tar (2-20 per cent), naphthalan oil (5-50 per cent), etc. Vaseline, lanolin, lard and other fats, as well as napthalan, are used as a fatty base for ointments.

9. *Compresses.* Hot compresses exert still deeper action than do ointments. A folded (2-3 layers) piece of cloth or gauze is moistened with water or some medicinal solution, placed on the skin, covered with a piece of wax-paper or oilskin, topped by a heavy layer of cotton and bandaged. Hot compresses are relatively rarely used in the treatment of skin diseases because the moist heat of a compress loosens the surrounding epidermis too much and may lead to irritation of the skin and concurrent pyogenic infection.

Ointment compresses—application to the skin of a heavy layer of ointment covered with wax-paper and cotton—are used more commonly.

10. *Plaster.* A heavy ointment closely adhering to the skin is called a plaster. Plasters are made of a special plaster material with an addition of various medicinal substances—ichthyol, mercury, salicylic acid, etc. Plasters exert still deeper action than do ointments because they are impermeable to water and prevent evaporation of moisture.

For better adhesion plasters are slightly heated over an alcohol burner or an electric stove before application. The skin is rubbed down with alcohol or benzene beforehand.

11. *Baths.* General and local baths are often prescribed for skin diseases. They are used as hydrothermal therapy (35-37°C) in such diseases as psoriasis, erythroderma and ichthyosis. Such baths act as sedatives and tonics on the nervous system, soften the skin, remove scales and promote better circulation and nourishment of the skin. If the patient has exudative and erosive sur-

faces, potassium permanganate is added to the bath as a disinfectant. In cases of abundant scaling soda (0.5-1 kg) is added to the bath. Tar baths are indicated in pruritic skin diseases, the tar either being added to the bath together with soap alcohol and water (100 g of tar, 75 g of soap alcohol and water each) or rubbed into the foci of affection, after which the patient takes the bath. In pruritic skin diseases good effects are also produced by baths with an oak bark decoction (1 kg of bark is boiled in 6 1 of water and the resultant decoction is added to the bath) or bran decoction (1 kg of wheat bran is boiled in 2-3 1 of water and the decoction is added to the bath).

Local baths (37-40°C) are prescribed for the treatment of skin diseases in which the processes are localised on the arms, legs, genitals and anal region. Potassium permanganate, bran decoction or other agents are often added to the baths.

The action of external agents on the skin depends not only on the medicinal form, but also on the character of the medicinal substance. For example, an indifferent zinc ointment exerts more superficial and delicate action than does a 10 per cent tar paste, although ointments generally possess deeper and more vigorous action than do pastes.

Substances possessing vigorous activity—tar, chrysarobin, resorcinol, etc.—are often used in the external treatment of patients affected with skin diseases. To prevent undesirable irritation of the skin and aggravation of the inflammatory process, such medicinal substances must at first be prescribed in weak concentrations. The concentrations are gradually increased. For example, in the treatment of eczema tar preparations are at first prescribed in the form of 1-2 per cent pastes and ointments. If the preparations are well tolerated, the concentration of tar is gradually increased to 5-10 and even 20 per cent.

Successful external treatment of skin diseases requires proper techniques of applying external agents. Improper techniques of external treatment aggravate, rather than improve, the inflammatory process.

Physiotherapy of Skin Diseases

Physical methods of treatment are often used in skin diseases.

Treatment with heat is indicated during various stages of the inflammatory process, especially during the subacute and chronic stages. An increased flow of blood caused by the action of heat improves the nutrition of the tissues and helps in resorption of the inflammatory infiltrate.

The heat applications include moist heat (baths and hot compresses) and dry heat (hot water bottles, Minin's blue lamp, sun lamp). Applications of paraffin and ozocerite exert vigorous and deep thermal action on the skin (see Supplement, p. 332).

Treatment with cold is administered in cases of acute inflammatory processes in the skin, hyperemia, edema and exudation. Cold is prescribed in the form of cooling, especially ice lotions; a bag with ice or snow is applied to the affected area less frequently.

Cryotherapy—refrigeration with solid carbon dioxide—is a special form of treatment with cold. Carbon dioxide released from a cylinder is transformed into snow—solid carbon dioxide—which is gathered in special tubes with pistons and is applied under pressure to the focus of affection for a period varying from 5 seconds to 1-2 minutes. During this period the affected area is refrigerated to a certain depth. After thawing the refrigerated tissues undergo necrosis. Cryotherapy is administered when it is necessary to destroy pathologic tissue in lupus erythematosus, birthmarks, acne rosacea, etc.

Ultraviolet rays constitute one of the sources of radiant energy particularly frequently used in the treatment of skin diseases. Exposure to ultraviolet rays (mercury vapour lamp) may be general or local. General exposure is usually resorted to daily or every other day. A course of treatment consists of 20-30 exposures. Doses producing erythema are used for local irradiation. The successive sessions are conducted only after disappearance of the inflammatory phenomena caused by preceding exposures.

Very important in the treatment of skin diseases is exposure to the sun, mention of which was made in the chapter devoted to problems of skin hygiene.

Roentgen rays, grenz rays, radium and radioactive isotopes are used in the treatment of skin diseases. Roentgen rays exert vigorous action in a number of skin diseases. Depending on the dose and method of their application they produce an anti-inflammatory effect, promote resorption of skin infiltrates, or cause epilation. Larger doses of these rays injure cells and lead to development of dermatitides, ulcers and atrophy of the skin.

Young cells and cells undergoing division are particularly sensitive to the action of roentgen rays. This circumstance is utilised for roentgenotherapy of malignant and certain benign neoplasms of the skin.

Grenz rays, or Bucky's rays, are similar to roentgen rays but have a longer wave and are retained in the superficial layers of the skin. They are often useful in eczema, neurodermatitides and other skin diseases.

Electrical energy is one of the forms of physiotherapy very widely used in the treatment of skin diseases. This includes low tension currents—galvanic current, iontophoresis and galvanocautery—and high tension currents—D'Arsonval current and diathermy.

Iontophoresis is the method of introducing medicinal substances into the organism by means of galvanic current. Iontophoresis

with solutions of potassium iodide, calcium chloride and novo-cain is most commonly used in the treatment of skin diseases.

Electrolysis is the use of galvanic current with pin-shaped electrodes to destroy warts, vascular birthmarks and excessive hair on women's faces. Electrolysis leaves delicate superficial scars.

Galvanocautery is a method of destroying pathologic tissue. The electrode is made in the form of a platinum loop which be-comes red-hot under the influence of galvanic current and cau-terises the tissues.

Diathermy is a high tension (up to 300 V) and high frequency electric current. A large amount of heat is produced in the tissues at the points of application of electrodes; the heat stimulates resorption of infiltrates and improves tissue nutrition.

Indirect diathermy—application of diathermic current to in-tervertebral nerve ganglions (cervical, thoracic and lumbar)—is widely used in the treatment of skin diseases.

Diathermy with pin-shaped electrodes is used for destruction of pathologic tissue in certain forms of tuberculosis of the skin, warts, birthmarks, excessive hairiness and other diseases (diathermo-coagulation).

D'Arsonval current is also characterised by high tension and high frequency. General and local d'arsonvalisation are dis-tinguished. General d'arsonvalisation is used in skin diseases involv-ing disturbances in the function of the central nervous system and in metabolism. Local d'arsonvalisation is useful in pruritic skin diseases. The use of pin-shaped electrodes also makes it possible to employ this current for destruction of pathologic tissue.

Ultrahigh frequency currents (UHF) are also used in the treatment of deep forms of pyoderma (furuncles, carbuncles, hidr-adenitides and ulcers). Indirect UHF therapy is effectively used in diffuse skin diseases. Administration of physiotherapy in skin diseases requires caution. Physiotherapy may be prescribed only by a physician and must be administered only by specially trained intermediate medical personnel.

PYODERMAS

Pyodermas are some of the most widespread skin diseases. They are caused by pyogenic cocci—staphylococci and streptococci (Figs. 13 and 14). Pyogenic cocci are widely distributed in the external environment. Large numbers of them are found on the surrounding objects, in the dust and in the air. That is why pyogenic cocci easily come in contact with the human skin. Their number on the surface of the body depends on the person's cleanliness. There is an average of about 40,000 microbes per 1 cm^2 of the skin; when man takes a bath he washes off 85,000,000-200,000,000 microbes many of which are pyogenic cocci. Staphylococci occur on the skin particularly frequently. The more often man washes and changes his underwear, the fewer pyogenic cocci there are on his skin.

However, development of pyoderma depends not only on the number of pyogenic cocci on the surface of the skin and their pathogenicity, i.e., capacity to produce disease. Of still greater importance is the general condition of the organism and its ability to resist the penetration of pyogenic cocci into the skin and their reproduction. M. Petrova has demonstrated by experiments on dogs that dysfunction of the nervous system ("nervous breakdown") may lead to development of furunculosis.

In man pyodermas may develop after psychic trauma, infectious disease, sharp cooling of the body, and other factors which weaken the organism. Various internal diseases also favour development of pyodermas. For example, stubborn furunculosis is very often observed in cases of diabetes mellitus.

Development of pyodermas also largely depends on conditions of external environment. The organism is adversely affected and development of pyodermas is stimulated by: (a) mechanical disruption of the continuity of the skin which facilitates penetration of pyogenic cocci into its deep layers; (b) irritation of the skin by various chemical substances, and (c) unfavourable fluctuations in the external temperature—long and systematic overheating which causes increased perspiration and maceration of the epidermis, and overcooling, especially oft-recurring and prolonged.

Unfavourable occupational influences are particularly important. In some shops workers often sustain small or even minute injuries by metal shavings, sawdust, scraps of tin, defective handles of tools, etc.
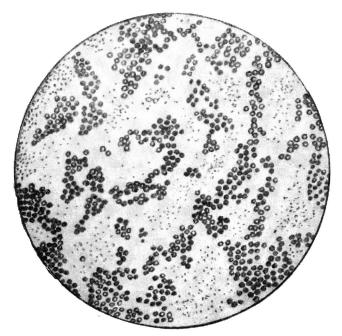
Fig. 13. Staphylococci (from A. Kartamyshev)
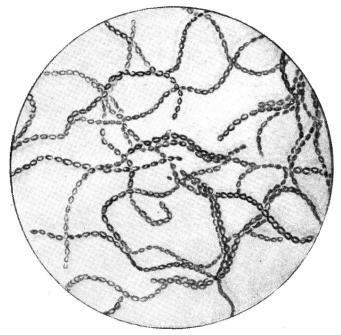
Fig. 14. Streptococci (from A. Kartamyshev)

Some chemical substances favour development of pyodermas if no adequate preventive measures are taken.

Mineral oils—products of petroleum refining and coal processing—come in contact with the workers' skin, penetrate into follicles and produce a chronic and sluggish inflammatory process—oil folliculitis which often favours penetration of staphylococci and development of furuncles.

Occupational dust also favours development of pyoderma because minute dust particles stop up the ostia of sebaceous and hair follicles and disturb their normal activity.

Staphylococcal and streptococcal pyodermas are distinguished according to the causative agent. Staphylococcal forms of pyoderma are characterised by affection of sebaceous and hair follicles. There are also mixed forms of pyoderma simultaneously produced by both species of pyogenic cocci. For practical purposes it is more important and convenient to distinguish pyodermas according to their clinical picture as *superficial* and *deep*.

In superficial forms of pyoderma the inflammatory process involves mainly the epidermis and but partly the derma. On healing these forms of the disease in most cases leave no trace except temporary pigmentation.

Deep forms of pyoderma are characterised by localisation of the infiltrate in the derma or in the subcutaneous adipose tissue. In retrograde development this infiltrate often undergoes purulent dissolution and is replaced by scar tissue.

Superficial pyodermas include impetigo, folliculitis and sycosis.

Impetigo, or purulent eruption, exists in three clinical forms.

1. *Streptococcal impetigo.* This disease usually affects the exposed parts of the body, most commonly the face, and frequently develops in people with a thin and delicate skin, especially in children. In debilitated children who have survived some emaciating disease streptococcal impetigo occurs more frequently and runs a more stubborn course. The disease affects women more often than men.

The disease begins with appearance of flat vesicles varying in size from that of a grain to that of a pea and containing a serous fluid. The vesicles rapidly grow larger, their contents very soon become turbid—seropurulent, and the vesicles transformed into pustules. The walls of the pustules are thin and flaccid and easily break especially on scratching. The pustules are often surrounded by a narrow inflammatory areola. Within only 1-2 days the pustules dry up and are transformed into thin, yellow, foliaceous crusts. A bright-red superficial erosion with a seropurulent discharge forms at the site of the ruptured pustules and quickly dries up into an amber-coloured crust. The crusts usually persist for about a week and are cast off, leaving a purplish macule which

soon pales and disappears. The eruption of pustules and formation of crusts are accompanied by moderate itching and tension of the skin, which makes the patients, especially children, scratch their skin. During scratching the purulent discharge comes in contact with the surrounding skin and infects it with pathogenic streptococci so that new pustules continuously appear and, if no proper treatment is administered, the process may persist for many weeks.

Streptococcal impetigo is a very contagious disease. Under conditions of close contact, for example, in the family or groups of children, it can be transmitted from one child to another. For want of control and in unhygienic surroundings streptococcal impetigo may affect masses of people.

There are certain varieties of streptococcal impetigo, which are of considerable practical importance.

(a) *Perlechè*. In the corners of the mouth streptococcal impetigo may run a protracted, chronic course. This is due to continuous irritation of the corners of the mouth by saliva and food, and to stretching by eating and talking. The disease begins with formation of a flat, oval vesicle in one or both corners of the mouth. The vesicle is quickly transformed into a pustule and then dries into a crust. A superficial erosion or a deeper fissure long persists under the dark, sanguineous crust.

This variety of streptococcal impetigo is very contagious. The disease is easily transmitted by kissing and using common towels and dishes.

(b) *Streptococcal paronychia*. On the terminal phalanges streptococcal impetigo sometimes assumes the form of a flat, semilunar pustule surrounding the nail. The pustule may open and be transformed into a superficial bright-red erosion surrounded by a border of exfoliating epidermis. In some cases this disease runs a stubborn, chronic course, affecting one finger after another.

There have been cases when contraction of streptococcal paronychia by some member of a nursery personnel caused a mass outbreak of streptococcal impetigo among the nursery children.

2. *Mixed impetigo* or *impetigo vulgaris*. This is a superficial pyoderma caused by mixed infection—streptococcal and staphylococcal (Fig. 15).

Streptococcal impetigo is very often concurrent with staphylococcal infection which changes the picture of the disease: the content of the vesicles soon becomes purulent, the crusts become thick and massive, and assume a grey or brownish colour due to admixture of blood. The crusts appear uneven and seem to be made up of crumbs ("crumby crusts"). They persist for 7-8 days and fall off leaving pigmented spots. Mixed impetigo greatly resembles streptococcal impetigo in that it is also contagious, is most commonly also localised on the face and other exposed parts of the body, and is predominant among children.
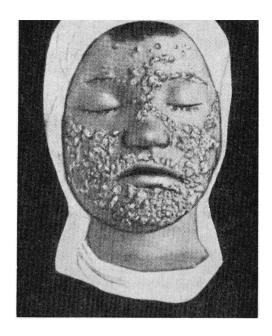
Fig. 15. Impetigo vulgaris

Impetigo vulgaris is quite often accompanied by a rise in temperature and enlargement of the closest lymph nodes.

Mixed impetigo may be a complication of another skin disease. For example, scabies is not infrequently complicated by impetigo vulgaris of the fingers and wrists, and subsequently of other parts of the skin. Impetigo vulgaris is a very frequent complication of pediculosis capitis.

3. *Staphylococcal impetigo* or *impetigo follicularis.* Semispherical or conical, tense pustules varying in size from that of a millet seed to that of a pea and containing thick yellowish or greenish pus form on the parts of the body covered with long or downy hair. The pustules are surrounded by a narrow areola which indicates the acutely inflammatory character of the disease. In the centre of each pustule there is usually a hair that projects through its cover. Staphylococcal impetigo is a superficial pustule at the ostium of a hair follicle, for which reason it is also called impetigo follicularis (Fig. 16).

The pustules of staphylococcal impetigo rarely burst and for the most part dry up, without opening, into compact brownish

Fig. 16. Impetigo follicularis (from A. Zenin
and N. Torsuyev)
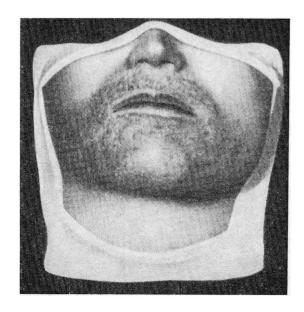
Fig. 17. Sycosis vulgaris

or yellowish crusts. The crusts persist for several days and then fall off leaving a temporary pigmentation.

Staphylococcal impetigo is most commonly localised on extensor surfaces of the extremities, but may appear on any part of the body.

Staphylococcal impetigo is less contagious than the two foregoing forms of impetigo.

4. *Folliculitis.* Folliculitis is an acute inflammatory process in a sebaceous and hair follicle caused by the staphylococcus. The process usually begins with affection of the ostium of the follicle, but due to low resistance of the organism, unfavourable external influences (trauma, etc.) and increased virulence of the staphylococcus, it subsequently involves deeper portions of the hair follicle, including the hair bulb.

Clinically folliculitis appears as a conical or semispherical pustule with its centre pierced by a hair. The pustule has an infiltrate at its base and is elevated above the level of the surrounding skin. The size of the infiltrate varies from that of a lentile to that of a pea, and the size of the pustule varies from that of a pinhead to that of a hemp seed.

Deeper folliculitides may upon healing leave small superficial scars.

5. *Sycosis.* A chronic eruption of folliculitides of the hairy parts of the face and body is called simple or nonparasitic sycosis (Fig. 17). Men most commonly have sycosis of the beard and mustache, and less frequently of the eyebrows, eyelids, armpits and pubes. Sycosis extremely rarely affects women. Like ordinary folliculitis sycosis is caused by the staphylococcus, but changes in the reactivity of the organism are the most important factor in this disease.

A. Pospelov held the cause of sycosis to be a disturbance in innervation of the hair follicles. Other investigators discovered in sycosis patients endocrine disturbances, especially hypofunction of the gonads. In some sycosis patients the disease is preceded by protracted rhinitis complicated by folliculitis of the nasal passages. Successful treatment of chronic rhinitis and folliculitis of the nasal passages expedites and facilitates the treatment of sycosis. The incidence of sycosis and the relapses of this disease are in many cases associated with failure to observe the rules of facial hygiene and with shaving under unsanitary conditions.

Superficial folliculitides appear in the affected area (most commonly on the hairy parts of the face) and their number rapidly increases. Deep folliculitides are gradually added. Many of these lesions coalesce and form infiltrated patches. In addition to the dried and crust-covered pustules new superficial and deep folliculitides appear. The skin in the affected areas becomes edematous and infiltrated (Figs. 18a and 18b). The hair in these areas is easily
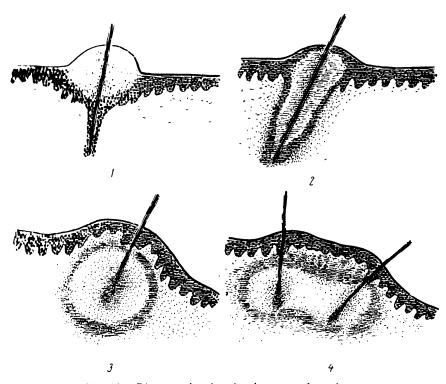
Fig. 18a. Diagram showing development of pyoderma
1—impetigo follicularis; 2—folliculitis; 3—furuncle; 4—carbuncle (from A. Zenin and
N. Torsuyev)
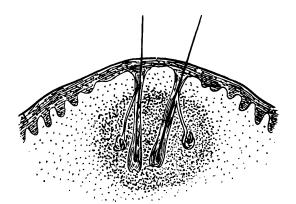
Fig. 18b. Diagram of hidradenitis (from A. Zenin
and N. Torsuyev)

epilated; sometimes it falls out by itself. The patients complain of itching, burning, contraction of the skin and pain.

Sycosis is characterised by a chronic course and a tendency to relapses.

Epidemic pemphigus neonatorum. This disease occurs in infants during the first weeks of life. It may assume the character of an epidemic outbreak in the children's division of a maternity home and in homes for infants. At a later age epidemic pemphigus usually develops in debilitated and untidily kept children.

Large, flaccid vesicles arise all over the child's skin and are later transformed into pustules with a seropurulent content. The pustules may grow to the size of a plum, a chicken egg and even larger. They either dry up and form crusts or open up and leave erosions.

Epidemic pemphigus greatly affects the child's general condition; the child becomes restless, often cries, sleeps badly and may have a poor appetite. In debilitated children epidemic pemphigus is often accompanied by fever and may develop into sepsis.

Deep pyoderma. Deep pyodermas include furuncles and furunculosis, carbuncles, hidradenitis, multiple abscesses in children and common ecthyma.

Furuncle and furunculosis. A furuncle, or boil, is an acute staphylococcal inflammation of the sebaceous and hair follicle and the surrounding tissues. The process begins with formation of a superficial folliculitis. Very soon the inflammation extends deeper into the hair follicle and then spreads to the adjacent tissues of the derma and subcutaneous adipose layer. A rather compact and not clearly defined infiltrate forms about the follicular pustule and involves not only the derma, but also the subcutaneous tissue. The skin in this area becomes red, then brown-red, and
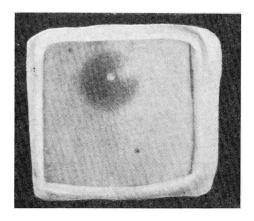
Fig. 19. Furuncle (from A. Kartamyshev)

Fig. 20. Furunculosis

develops edema. The patient complains of pulsating pain. The in-
filtrate gradually increases in size, reaching that of a plum, a
chicken egg and even larger. The edema and pain also increase.
Within a few days the central part of the infiltrate undergoes
necrosis and purulent dissolution. The central part of the furuncle
softens, the skin over it becomes thin and the furuncle opens and
discharges thick, yellowish-green pus. After opening, the centre
exhibits necrotic tissue about the hair follicle—the "necrotic core"
which usually detaches itself within a day or two, exposing a small
ulcerous defect which rather rapidly fills with granulations and
forms a scar. In most cases the development of a furuncle lasts a
total of 1-2 weeks (Fig. 19).

Furuncles may appear on any part of the skin about a hair
follicle. Most commonly they arise on the thighs, buttocks, neck,
forearms and face. Quite often the first furuncle is followed by
another, a third one, etc. The simultaneous existence of several
furuncles or the appearance of one furuncle after another is called
furunculosis (Fig. 20).

Furunculosis may be local if the furuncles are concentrated on
a circumscribed part of the skin. Local furunculosis is almost al-
ways associated with the action of external irritating factors.
Local furunculosis on the back of the neck occurs comparatively
frequently in men and is due to friction of the collar and the pop-
ular custom of shaving the neck, which causes numerous minute

disruptions of the continuity of the epidermis thereby facilitating the penetration of staphylococci into the skin.

If the furuncles are disseminated over various parts of the skin, the furunculosis is called *general*.

Furuncles and furunculosis in some measure affect the general condition of the organism. Fever is often observed, especially in furunculosis. An accutely inflamed and painful swelling of the adjacent lymph node can always be seen, and lymphangitis can sometimes be observed. The intense pain often deprives the patient of sleep and causes temporary disability.

Furuncles on the upper lip, nose and other parts of the face are particularly dangerous because of the threatened complication with sepsis.

Furunculosis may persist for months. In some patients it recurs stubbornly over a period of years. Chronic furunculosis is always based on considerable disturbances in the general condition of the organism, namely, dysfunction of the central nervous system, metabolic disorders, extreme emaciation, etc.

Furunculosis may have a secondary origin, i.e., it may arise as a result of scratching in patients affected with scabies and other pruritic skin diseases.

A *carbuncle* is also caused by a staphylococcus and is a simultaneous affection of several, closely located sebaceous and hair follicles. The infiltrates forming in a carbuncle merge into a single, large node which reaches the size of a chicken egg, a first, and even larger. Thus carbuncles form as the result of several furuncles coalescing into one continuous infiltrate.

The affected area is purplish-brown with several follicular pustules in the central part. It is hard and elevated above the level of the surrounding skin which is edematous. In most cases it is accompanied by sharp pain, headache and fever.

Subsequently the purulent infiltrate opens, abundant sanguineous pus is discharged and necrotic cores detach themselves. A considerable ulcerous defect is formed; it gradually fills with granulations and heals leaving a scarred excavation.

In most cases the carbuncular process lasts 3-4 weeks. Carbuncles often run a very severe course with vast necrosis, high temperature, chills, unconsciousness and phenomena of sepsis. Lethal results are possible.

Carbuncles most commonly occur in people with lowered resistance—elderly, emaciated and diabetic.

Hidradenitis is an acute, inflammatory, staphylococcal disease of the apocrine sweat glands. In man apocrine sweat glands are found mainly in the axillas, and hidradenitis is localised predominantly in these areas (Fig. 21). It is observed in sexually mature people, most commonly in women. The disease begins with the appearance of a somewhat painful node, the size of a
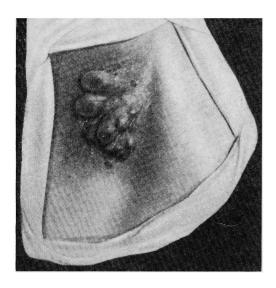
Fig. 21. Hidradenitis

lentile or pea, deep in the subcutaneous tissue. The node gradually enlarges and grows more painful. Within a few days the node becomes as large as a cherry or even a walnut and adheres to the derma. The skin over it grows red, purplish-red, and a fluctuation appears. The infiltrate undergoes necrosis and suppuration, and is transformed into an abscess. The pain often becomes so intense that movements in the shoulder joint become almost impossible. Then the abscess opens at one or several points from which copious creamlike pus is discharged. The cavity of the abscess is gradually emptied through these openings, the pain diminishes and the infiltrate begins to be resorbed. The development of hidradenitis takes a total of about 2-3 weeks. Very often several nodes of different sizes form at the different stages of the disease. If the node keep recurring, the process may last many weeks.

Hidradenitis may very unfavourably affect the patient's general condition. Excessive sweating, pruritic skin diseases, especially in the axillary region, work under conditions of high external temperature and occupational dust are conducive to development of the disease.

Multiple abscesses in children. Infants, especially those who perspire freely because of excessive wrapping, those who are untidily kept and those who are affected with dyspepsia sometimes
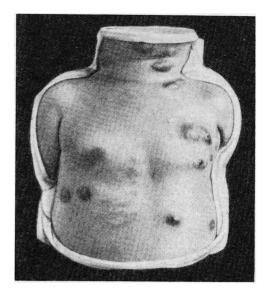
Fig. 22. Multiple abscesses in children
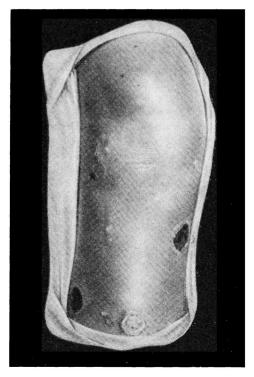
Fig. 23. Ecthyma vulgaris

have a peculiar pyoderma—multiple abscesses, or pseudofurun-
culosis. These abscesses develop as the result of penetration of
staphylococci into the ostia of sweat glands (eccrine).

In sweat glands abscesses develop at different depths. If the
process is confined to the ostium of the duct of the gland, a pustule
simulating staphylococcal impetigo, but having no hair in the
centre, is formed.

In cases of affection of deeper parts of the gland numerous
infiltrated papules and nodes are formed in the derma or the sub-
cutaneous tissue. They gradually increase in size from that of a
hemp seed to that of a plum, are quite hard and of a saturated-
red or purplish-brown colour (Fig. 22). Subsequently they begin
to soften and finally open at one or several points where they dis-
charge a large amount of thick, yellow-greenish pus. The nodes
simulate furuncles, but differ from them by the absence of a hair-
perforated pustule at the top of the node. No necrotic core is formed.

The disease may last several weeks. The intense pain in the
nodes makes the child restless and causes sleep and appetite dis-
turbances. In debilitatéd children multiple abscesses may become
aggravated by sepsis.

Echtyma vulgaris. This disease is caused by a streptococcus.
It is usually observed in emaciated, debilitated and anemic peo-
ple. Flaccid, flabby pustules with seropurulent contents appear
on the shanks, and less frequently at other sites. The pustules
vary in size from that of a lentile to that of a bean. They are sur-
rounded by an areola of a purplish-red, rather dense infiltrate and
somewhat simulate streptococcal impetigo, but differ from it in
that the pustules are always located deep in the derma and have
a thicker and therefore stronger covering (Fig. 23). Within 2-3 days
the pustules dry up and are transformed into thick yellowish or
sanguineous crusts covering shallow ulcers which have a red floor
coated with a purulent discharge. Ecthyma is a chronic disease.
In the absence of proper treatment the ulcers under the crusts
may disintegrate over a period of weeks and even months. With
correct treatment ecthyma can be cured in 3-4 weeks.

The floor of the ulcers is gradually cleansed and filled with
granulations. Upon healing ecthyma always leaves a scar.

In ecthyma vulgaris the number of pustules rarely exceeds
one or two dozens. Often there are only single pustules.

Chronic atypical pyoderma. Chronic atypical pyoderma occu-
pies a special place among pyodermas and must be distinguished
as a separate group of purulent skin affections. These affections
occur comparatively less frequently than do superficial or deep
pyodermas, but are very important because they run a protracted
and stubborn course and are difficult to treat.

The group of chronic pyodermas includes several clinical forms
which greatly differ in their manifestations. They all run a chronic

Fig. 24 Chronic ulcerative pyoderma
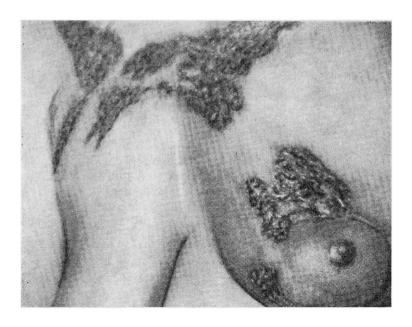
Fig. 25. Chronic ulcerative-vegetative pyoderma

course, have a tendency to relapses and aggravations, and resist treatment.

Chronic ulcerative and chronic ulcerative-vegetative pyodermas are the most common forms. The patients affected with these forms of pyoderma develop painful and irregularly shaped ulcers which discharge a large amount of pus. The ulcers differ in size and depth, and run an extremely protracted course (Fig. 24). Sometimes the floors of the ulcers are covered with papillae; pressure on the papillae results in a discharge of numerous droplets of pus between them ("the sieve symptom') (Fig. 25).

This course of pyoderma is based on deep changes in the reactivity of the organism. Chronic pyoderma patients exhibit certain disturbances in the function of the nervous system, endocrine disorders, vitamin C deficiency and other important deviations. Some chronic pyoderma patients show an unusual reaction at the site of intracutaneous administration of 0.1 ml of staphylococcus vaccine; in certain cases the reaction is very violent with vast redness, edema and even necrosis in the centre (hyperergy, sharply increased reactivity); in other cases there is, on the contrary, a complete absence of any visible reaction (anergy, sharply diminished reactivity). Histologically the manifestations of chronic pyoderma in most cases exhibit the structure of infectious granuloma which is a circumscribed nodular proliferation of granulation tissue with a special structure of the cells.

Chronic ulcerative-vegetative pyoderma is one of the most frequently occurring forms of chronic pyoderma. It is characterised by formation of ulcers of various sizes and shapes with soft, infiltrated edges, sometimes eroded and hanging over the floor (see Fig. 24). The floor and edges of the ulcers may be covered with papillae (see Fig. 25). The ulcers are formed as a result of softening and disintegration of the compact nodes localised in the subcutaneous cellular tissue. Ulcerative-vegetative pyoderma arises no less frequently at the sites of usual pyodermas—furuncles, ecthyma, folliculitides and even impetigo.

TREATMENT OF PYODERMAS

Pyodermas are skin diseases for most of which external treatment alone does not suffice. This stands to reason, considering the role played in the development of pyodermas by the lowered resistance of the organism, infections and dysfunction of the nervous system and the internal organs. In addition to local treatment most patients must also be prescribed general treatment.

General Treatment of Pyodermas

Antibacterial preparations (antibiotics and sulfonamides), immunobiological methods which increase the resistance of the organism to pyogenic cocci, and general roborants are used in the general treatment of pyodermas.

I. **Antibiotics** play an important part in the treatment of pyoderma patients. The depressive action of penicillin and other antibiotics on pyogenic cocci leads to rapid diminution in the infiltrate, the abatement of pain, cessation of new eruptions, and healing of erosions or ulcers.

Owing to the treatment with antibiotics it is often possible to avoid purulent dissolution of the infiltrate in furuncles, carbuncles, hidradenitides and multiple abscesses in children, and "dry" resorption of the infiltrate takes place. Administration of antibiotics expedites restoration of the working capacity of pyoderma patients and is therefore particularly useful in deep forms of pyoderma. Antibiotics are also used in the treatment of some forms of superficial pyoderma—sycosis, recurrent impetigo and folliculitides—and chronic atypical pyoderma.

However, treatment with antibiotics does not prevent relapses of pyodermas because antibiotics exert a comparatively weak influence on the processes of immunity. Antibiotics must therefore always be prescribed together with other therapeutic agents.

In recent years antibiotics have been observed to be less effective in the treatment of staphylococcal forms of pyoderma and of furuncles, carbuncles, hidradenitis, impetigo neonatorum, and multiple abscesses in children. The reason for it is that the staphylococcus is a microbe in large measure capable of adapting itself to changing conditions of existence. The staphylococci isolated from the pus of pyoderma patients increasingly more often prove to be insensitive or but scarcely sensitive to penicillin and other antibiotics. It has been observed that the more extensively some antibiotic is used in a given group of the population or in a certain area, the more frequently the causative agents are staphylococci insensitive to this antibiotic. It is therefore very desirable to determine beforehand the sensitivity of the staphylococci isolated from the patient's pus to various antibiotics and to prescribe the antibiotic to which the causative agent happens to be sensitive.

Another very important method of enhancing the therapeutic effects of antibiotics is simultaneous administration of immunisation therapy to patients with staphylococcal pyodermas.

1. *Penicillin.* Ecmonovocillin (procaine penicillin with ecmolin) in a dose of 600,000 u intramuscularly once a day or bicillin in a dose of 1,200,000 u once in 4-5 days are the best penicillin preparations to be administered to out-patients. In-patients are

given either ecmonovocillin or penicillin in physiologic solution in a dose of 50,000 u every 3 hours.

In the treatment of pyodermas penicillin may also be administered in a dose of 150,000 u in 1.5 ml of a 0.5-1 per cent novocain solution 4 times a day.

The total dose of penicillin depends on the clinical form of the pyoderma. Doses of about 2,000,000 u are used for the treatment of single furuncles. The treatment of furunculosis, a carbuncle and hidradenitis requires doses of 2,000,000-3,000,000 u and more. In the treatment of multiple abscesses in children penicillin is administered in a dose of 50,000 u in 1 ml of a 0.5 per cent novocain solution 4 times a day (a total of 1,000,000 u). In sycosis and chronic atypical pycderma the dose is 4,000,000-5,000,000 u and even higher.

2. *Treatment with other antibiotics.* Streptomycin is of little effect in pyodermas. It is used only when penicillin proves ineffective or when a patient has simultaneously tuberculosis and pyoderma. The dose is 0.5 g per day.

Synthomycin, levomycetin or *chloromycetin* (varieties of chloramphenicol) are prescribed mainly for patients with furuncles, hidradenitis and sycosis and are administered per os in a dose of 0.5 g 3-4 times a day (total dose—15-25 g).

Chlortetracycline, tetracycline and *terramycin (oxytetracycline)* are also administered per os in a dose of 0.2 g 4-5 times per day, 5-15 g per course of treatment, depending on the effects and tolerance of the treatment. They are useful in sycosis, furunculosis, hidradenitis and chronic pyoderma. *Erythromycin* is also helpful in the treatment of pyoderma; this preparation is administered per os in a dose of 0.5 g 3-4 times a day (a total of 7-15 g).

II. **Sulfonamides**. In the treatment of pyodermas sulfonamides are inferior to antibiotics. Norsulfazole (sulfathiazole), ethazole (2[para-aminobenzol-sulfamido]-2-ethyl-3,4-thiadiazole) and sulfadimesine (sulfamezathine) are given per os in a dose of 1 g 3-4 times a day for 5-10 days mainly for the treatment of deep forms of pyoderma. If the disease exhibits a tendency to relapses, sulfonamides cannot prevent them.

III. **Immunobiological methods of treatment**. Most pyoderma patients do not develop stable immunity. The absence of marked immunity after an attack of pyoderma is one of the main reasons for the relapses of this disease. To strengthen the organism in its struggle against pyogenic infection, to intensify the processes of immunity and prevent relapses of the disease, immunobiological methods of treating pyoderma are used. Specific and nonspecific immunotherapy is distinguished.

(a) *Specific immunotherapy.* For specific immunotherapy various bacteriological preparations of staphylococcus cultures are used

Staphylococcus anatoxin is the most commonly used preparation. It is administered subcutaneously into the interscapular region in gradually increasing doses of 0.2-0.5-1-1.5 and 2 ml. In the beginning of the treatment the intervals between injections are 2-3 days; during the later half of the treatment the intervals are 4-5 days, depending on the reaction to the preceding injection. The total reaction (rise in temperature, headache), local reaction (pain at the point of injection) and focal reaction (increased redness, swelling and pain in the foci of pyoderma) are taken into account. The course of treatment consists of 6-10 injections.

Other staphylococcus preparations—staphylococcus filtrate, vaccine, antiphagin and phage—are also used for the treatment of pyodermas.

Specific immunotherapy is indicated in furunculosis, hidradenitis, and sometimes in folliculitides, sycosis and chronic pyoderma. This type of treatment, although inferior to antibiotics as regards rapidity of action, is superior in its influence on the processes of immunity: the patient's phagocytosis improves and the content of antibodies in the serum increases. Antibiotics and immunotherapy seem to supplement one another and are therefore prescribed simultaneously.

(b) *Nonspecific immunotherapy.* Certain nonspecific immuno-biological methods of treatment—autohemotherapy, lactotherapy and injections of laky blood—also help to increase the immunity to pyogenic cocci. Injections of the mother's or donor's blood are useful in multiple abscesses in children; the injections begin with 1-2 ml and are gradually increased to 5-6 ml once in 3-4 days (a total of 5-8 injections).

IV. **General roborant treatment.** In the treatment of pyodermas an important part is played by methods aimed at strengthening the organism and increasing its resistance to pyogenic infection. In such cases it is necessary to be guided by the results of the patient's examination. For example, if the patient suffers from diabetic disturbances in carbohydrate metabolism, it is necessary to limit the amount of carbohydrates in the diet and sometimes to prescribe insulin. Anemia requires prescription of vitamin B_{12}, iron, arsenic, phosphorus (phytin) and campolon (aqueous liver extract). In cases of hypovitaminosis and avitaminosis the corresponding vitamins are prescribed. Intravenous injections of a 5 per cent ascorbic acid solution—10 ml daily for 15-20 days—are very useful in the treatment of ecthyma vulgaris, recurrent furunculosis and, especially, chronic pyoderma. Vitamin P is simultaneously administered per os. Vitamin A and riboflavin are additionally administered in folliculitides and furunculosis. It is also essential to establish a proper general regimen for the patient (adequate sleep and rest, fresh air) and eliminate all irritation of the focus of affection.

Local Treatment of Pyodermas

A proper hygienic regimen is a sine qua non of effective treatment. To prevent the pyogenic cocci from spreading over the skin washing is prohibited. Instead the skin around the suppurative lesions is rubbed down 2-3 times a day with a camphorated alcohol solution, or 2 per cent salicylic alcohol or 3 per cent synthomycin alcohol. The underwear must be changed at least once or twice a week. However, if the pyoderma lasts more than 1.5-2 weeks, the patient must be allowed to wash his body once a week; otherwise soiling of the skin may promote vigorous multiplication of pyogenic cocci on its surface and the appearance of new foci of the disease. To diminish the possibility of inseminating the skin with pyogenic cocci, it is good to wipe the skin with an alcoholic disinfectant after washing (see Supplement, p. 324).

Baths with potassium permanganate (1:20,000) every other day are prescribed for the treatment of multiple abscesses in children. This makes it possible to keep the child's body clean and stimulates resorption of the inflammatory nodes.

The patients' finger-nails must be trimmed short. It is recommended to paint the fingertips of impetiginous children once or twice a day with a 2 per cent iodine tincture.

In local treatment of streptococcal impetigo and impetigo vulgaris the crusts are first softened with oil and carefully removed, and the covers of the pustules, as well as the wall fragments of the already ruptured pustules, are cut away with scissors. Then all the erosions are painted with a 1 per cent methyl violet or brilliant green solution in 70 per cent alcohol. About 10 or 15 minutes later one of the disinfecting ointments is applied to the affected area. Good effects are produced by a 1 per cent synthomycin emulsion, and a 3 per cent synthomycin, a 3 per cent chlortetracycline, a 5 per cent colimycin (antibiotic of the neomycin group), and a 1-3 per cent oxytetracycline ointment. The following ointments are also used with some success: 2-3 per cent yellow mercuric oxide, 3-5 per cent ammoniated mercury, 5-10 per cent sulfur, 5 per cent xeroform or dermatol, liquid pitch and boric acid (10 per cent boric acid and 5 per cent liquid pitch), and penicillin (1,000 u per 1 g of the base). Good effects in pyoderma are also produced by an ointment containing polymyxin (10,000 u per 1 g of the base) and levomycetin or one of the antibiotics of the tetracycline series.

For the treatment of staphylococcal impetigo and folliculitis the affected areas are painted with methyl violet or brilliant green after first puncturing with a needle or cutting away with scissors the covers of the pustules and removal of the pus with a piece of cotton. Then one of the same disinfecting ointments is applied. Deep folliculitides are painted with pure ichthyol.

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One of the best agents for the treatment of sycosis vulgaris is a 1 per cent (or 10 per cent) synthomycin emulsion, or a 3 per cent synthomycin and a 3 per cent chlortetracycline ointment. Before applying these ointments it is necessary carefully to open with scissors the pustules, remove the pus with a piece of cotton and paint the affected area with a 1 per cent solution of methyl violet or brilliant green.

In cases of stubborn sycosis the hair in the affected areas must be removed by hand with the aid of an epilation forceps and the aforementioned external treatment must be continued. Good effects are produced in many cases a 10-50 per cent paste containing naphthalan oil.

The treatment of ecthyma vulgaris must begin with softening the crusts by means of a 3 per cent salicylic ointment and their removal. This is followed by a daily application to the ulcer of a heavy layer of a streptocide water (a brand of sulfonamide) paste or other sulfonamide. The streptocide paste is used for several days until the copious purulent discharge disappears, after which the following disinfecting ointments are prescribed: tetracycline-polymyxin ointment, a 5 per cent xeroform ointment, dermatol ointment, Vishnevsky's ointment and Mikulicz' ointment (see Supplement, pp. 324—325).

Favourable effects are produced in many cases of pyoderma by ointments containing antibiotics with a broad spectrum of action and corticosteroids.

A good effect is produced by a bandage made of strips of adhesive plaster applied shingle-manner for a period of 3-7 and even 10 days.

The main agent for the external treatment of furuncles, carbuncles, hidradenitis and multiple abscesses in children is pure ichthyol which is applied in the form of a flat cake (see Supplement, p. 333).

Disinfecting ointments are applied after emptying of the abscess cavity (hidradenitis, multiple abscesses in children) or detachment of the necrotic core (furuncle, carbuncle).

In cases of multiple abscesses in children the clearly pulsating large abscesses must be cut open and pure ichthyol must be applied; 1 or 2 days after this disinfecting ointments must be used.

Treatment of deep pyoderma with hot compresses is inexpedient because of the possible maceration and infection of the surrounding skin.

Local treatment of chronic pyoderma depends on its clinical picture. In ulcerative forms a shingle-like bandage of strips of adhesive plaster is applied for a period of 5-7 days. In vegetative forms good effects are produced by scraping out the vegetations with a sharp curet under local anesthesia with subsequent application of streptocide water paste.

Physiotherapy. Exposure to a mercury vapour lamp, ultrahigh frequency currents and a sun lamp is widely used in the treatment of pyodermas.

In many cases good effects are produced on pyoderma patients by general daily irradiation with a mercury vapour lamp (suberythema doses). Erythema and suberythema doses of ultraviolet rays are used for local irradiation. Such irradiation doses are indicated in the incipient stages of development of furuncles and hidradenitis. Every successive irradiation is given several days after disappearance of the erythema produced by the previous irradiation.

Ultrahigh frequency currents produce favourable effects in the treatment of furuncles and hidradenitis. Exposure to a sun lamp is indicated in cases of large and very painful furuncles and in hidradenitis.

Roentgen therapy may be administered in some cases of pyoderma, usually in hidradenitides and furuncles on the face, especially when antibiotics are ineffective or contraindicated. Roentgen therapy is administered in the incipient stages of hidradenitis or furuncles.

In most cases of sycosis vulgaris roentgen therapy results in but temporary improvement and does not prevent relapses of the disease. Sycosis recurring after roentgen therapy runs a more severe and stubborn course.

PREVENTION OF PYODERMAS

The pus discharged in pyodermas always contains tremendous numbers of pyogenic cocci. Large numbers of pyogenic cocci are also always found on the skin surrounding the purulent lesions, for which reason measures must be taken to safeguard the people around the patient against infection. The patient must have individual underwear, bed linens and towel which should be changed at least once or twice a week and disinfected in a disinfection chamber or by boiling with lye. The contaminated dressing material must be burned.

In the event a child contracts impetigo he must be prohibited from associating with children until cured.

Prevention of pyoderma in industry and agriculture requires particular attention. The principal aim is to safeguard the health of the workers and to prevent them from contracting pyoderma. Pyoderma often involves temporary disability. Strict observation of the rules of pyoderma prevention in industry and agriculture favours diminution in the incidence of pyoderma in these branches of the national economy. Proper organisation of pyoderma control in production is one of the most important tasks of medical workers.

The following measures are aimed at preventing pyoderma in production:

1. Sanitary-engineering measures diminishing or eliminating the effects of unfavourable factors.These include proper organisation of the working place, mechanisation of labour, and removal of metal shavings and dust by means of special hooks and brushes. Guard shields are installed to prevent the cooling oil or emulsion from spraying during work and splashing the workers.

2. Hygienic measures: adequate lighting, good ventilation and regular cleaning of the premises.

3. Immediate treatment of minor skin injuries. At first the skin around a minor injury is wiped off with a piece of cotton soaked in an alcohol-camphor solution, 1 per cent salicylic alcohol or benzene. Then the site of injury is painted 2-3 times at 1-2 minute intervals with a 1 per cent solution of methyl violet or brilliant green in 70 per cent alcohol or a 2-5 per cent iodine tincture. Small abrasions, scratches, fissures and superficial wounds may be painted with Novikov's solution (antiseptic preparation containing brilliant green, ethyl alcohol and castor oil) (see Supplement, p. 325).

This solution dries within 1.5-2 minutes and forms a dense film which protects the affected area from further contamination and irritation and acts as a disinfectant.

4. Individual protection of the workers' skin from contaminating and irritating substances. For this purpose the workers are given special work clothes—gloves, mittens, overalls, aprons, etc. The work clothes are put on immediately before work and are taken off right after the end of work. They must be strong and clean.

One of the foremost concerns of the medical workers employed at industrial enterprises is to see to it that the work clothes are washed, repaired and changed regularly.

Protective ointments and pastes (IER-1 and IER-2)* are sometimes also used to safeguard the skin against contamination during work. They are applied to the skin in a thin layer before the beginning of work and form a film which protects the skin from penetration of the irritating substance.

5. Observance of the rules of personal hygiene. It is well known that habitually clean people suffer from pyodermas much less frequently than do slovenly people.

In branches of industry and agriculture where the skin is soiled during work the workers must take shower baths at the end of work.

Cleansing of the skin from occupational contaminants is facilitated by the use of "washing pastes". After work these pastes

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* IER-1 is a paste containing soda soap, kaolin, glycerin and water. IER-2 is a paste containing paraffin, ceresin and vaseline.

are rubbed into the soiled skin and are washed off with warm water. Rakhmanov's paste is the one most commonly used (see Supplement, p. 333.).

Workers must be warned against washing their hands with sawdust, sand, pumice, kerosene, acetone and cooling emulsions since these substances easily injure the stratum corneum, the skin becomes dry and degreased, and pyogenic cocci readily penetrate into the skin where they produce pyodermas.

To prevent pyoderma among workers of the peat industry, I. Paikin has proposed a hand-scrubbing method (see Supplement, p. 333). This method is also useful for workers of other industries, especially in cases of dry skin, hyperkeratosis, callosities, fissures and minor injuries.

Health education plays an important part in the control of pyoderma.

FUNGUS DISEASES OF THE SKIN

Fungus diseases of the skin, or dermatomycoses, are caused by fungi—vegetable parasites. The structure of pathogenic fungi closely resembles many other fungi, growing in forests or on rotting objects, and saprophytes. In the process of evolution pathogenic fungi separated from the other fungi and adapted themselves to parasitising on the skin of man and animals.

Under the microscope pathogenic fungi appear as filaments with round, oval or irregularly shaped spores. The filaments are the mycelium, i.e., the "body" of the fungus, and the spores are the organ of reproduction.

Four groups of fungus diseases are distinguished: keratomycoses, epidermomycoses, trichomycoses and deep mycoses (A. Arievich's classification).

1. *Keratomycoses.* The fungi parasitise only on the stratum corneum of the epidermis and evoke no response inflammotory reaction of the skin. The diseases of this group are scarcely contagious and include pityriasis versicolor and erythrasma.

2. *Epidermomycoses.* The causative agents of this group of skin diseases also parasitise on the stratum corneum of the epidermis but their waste products possess irritating action and cause an inflammatory process in the skin. Moreover, these fungi are capable of parasitising on the nails and mucous membranes. This group of diseases includes dermatophytosis and candidid. The diseases of this group are not equally contagious.

3. *Trichomycoses.* The diseases of this group include trichophytosis, microsporosis and favus. These diseases are characterised by affecting the hairy part of the head. The smooth skin is also affected. Some diseases of this group also involve the nails. The causative agents of these diseases parasitise on the stratum corneum of the epidermis and evoke an inflammatory response reaction of the skin. The inflammatory phenomena are, as a rule, more strongly pronounced when the disease is caused by a fungus which usually parasitises on animals and less strongly marked when the causative agent parasitises only on man. Trichomycoses are very contagious.

4. *Deep mycoses.* Deep mycoses are characterised by affecting the deep layers of the skin—the derma and subcutaneous adipose tissue. The pathologic process caused by the fungi which produce these diseases often involves the internal organs, bones and nervous system. The deep mycoses include actinomycosis, maduromycosis (madura foot or mycetoma), blastomycosis (American
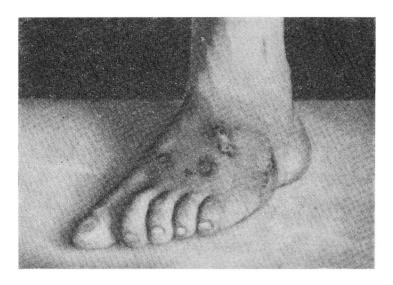
Fig. 25*a*. Deep mycosis (maduromycosis or mycetoma)
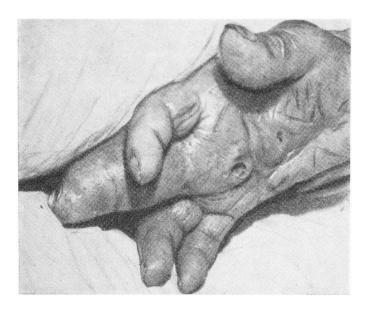
Fig. 25*b*. Deep mycosis (sporotrichosis)

and European), sporotrichosis, coccidioidomycosis, etc. The infection with deep mycoses occurs variously. In some cases the causative agent gains entrance into the skin when its continuity is disrupted by vegetable, wooden or other materials; in other cases it penetrates into the organism with dust through the respiratory tract; in still other cases the fungus may long exist as a saprophyte in the oral cavity or the intestines and under conditions unfavourable to the organism may acquire pathogenic properties and become the causative agent of deep mycosis. In these diseases various lesions—papules, vesicles, pustules, tubercles, vegetations and particularly often nodes and deep infiltrations—are formed on the skin (Figs. 25a and b). Disintegration and ulceration of these lesions lead to considerable disturbances in the patients' general condition, while affection of the internal organs, bones and nervous system not infrequently results in sepsis, intoxication and death.

The most important of the fungus diseases are the dermatomycoses which affect the hair, namely, trichophytosis, microsporosis, favus and epidermophytosis. These diseases are widespread and very contagious.

The clinical pictures of trichophytosis and microsporosis have similar features and the diseases are sometimes unified by the common designation of *tinea capitis*.

TRICHOPHYTOSIS

Trichophytosis is caused by various species of *Trichophytons*. There are *Trichophytons* which parasitise on the skin of man and animals. The *Trichophytons* parasitising on the human skin hardly ever affect animals, while those parasitising on the skin of animals may produce skin diseases in man.

The *Trichophytons* parasitising only on man always lodge, when affecting the hair, inside the hair shaft (*Trichophyton endothrix*. Fig. 26). Viewed under the microscope the hair affected with the fungus contains numerous large spores arranged in parallel chains along the axis of the hair. The *Trichophyton endothrix* affects the hair, smooth skin and nails. The fungi of this type cause *superficial trichophytosis*.

Superficial Trichophytosis of the Hairy Part of the Head

This form of trichophytosis occurs mainly in preschool- and schoolchildren, although infants are likewise often affected with this disease. With the onset of the period of sexual maturation the susceptibility of the human organism to the *Trichophyton endothrix* sharply diminishes. Superficial trichophytosis of the hairy part of the head after the age of 15 is very rarely observed.

The disease begins with formation of pinkish-reddish, round macules on the hairy part of the head. During the first week the macules grow larger and then their growth ceases. Very soon they become covered with whitish or greyish branny scales and the fungus rapidly invades the shaft of the hair. By growing into the hair the fungus destroys the substance of the latter and the hair
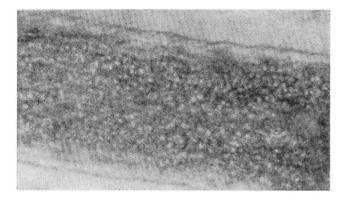
Fig. 26. Hair invaded by *Trichophyton endothrix* under the microscope (from A. Arievich and Z. Stepanishcheva)

becomes fragile. The hair shaft invaded by the fungus breaks at a height of 1-3 mm above the level of the skin. In the foci of affection the hair becomes thin because part of it breaks off (Fig. 27). Hair fragments ("stubs") standing on end, often whitish and sometimes appearing twisted, can be seen in the foci with thinned hair. The hair frequently breaks at the skin level, in which case it appears as "black dots". The hair fragments are often hard to see because of the scales covering the foci.

A microfocal variety of trichophytosis occurs most commonly. The foci are usually from 2-3 mm to 1 cm in diameter. Larger foci — 2-3 cm and more—are observed less frequently. The longer trichophytosis is left untreated, the more new foci are formed. Sometimes the foci of affection may disappear within a few months without treatment, but new foci appear next to them. In the absence of treatment trichophytosis runs a protracted, chronic course. With the onset of sexual maturity most trichophytosis cases heal spontaneously. Sexual maturation involves a deep reorganisation of the entire organism. The activity of the central nervous system, the functions of the endocrine glands, the metabolic processes and the secretion of the sebaceous glands change. As the result of these changes the conditions for the existence of *Trichophytons* in the skin and hair become unfavourable and their further development impossible.

Fig. 27. Superficial trichophytosis of the hairy
part of the head (from A. Arievich and
Z. Stepanishcheva)

Superficial trichophytosis of the beard and mustache occurs much more rarely than that of the hairy part of the head. This is due to the greater resistance of the adult organism to the *Trichophyton endothrix*. Trichophytosis of the beard and mustache is most frequently the result of infection by shaving with a contaminated and undisinfected razor, shaving brush, etc.

Superficial Trichophytosis of the Smooth Skin

Superficial trichophytosis of the smooth skin is either concurrent with superficial trichophytosis of the hairy part of the head or it may develop alone. Affection of the smooth skin begins with formation of a pink-red round or oval macule with clearly defined borders. The macule has a brighter peripheral part elevated in the form of a flat roll. The central part of the macule is covered with branny scales. The edges of the macule exhibit vesicles, the size of a pinhead, and crusts. The macule rapidly enlarges and its centre gradually pales and ceases to exfoliate (Figs. 28 and 29). It usually grows to 2-3 cm in diameter, sometimes larger. As the macule enlarges it becomes increasingly more annular. Sometimes some of the rings coalesce and form complex figures.

Trichophytosis of the smooth skin is usually observed on the exposed parts of the body.

In the absence of treatment superficial trichophytosis of the smooth skin may last several weeks and even months, after which the macules begin to disappear.
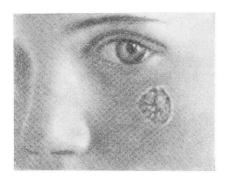
Fig. 28. Superficial trichophytosis of
the smooth skin (from A. Arievich
and Z. Stepanishcheva)
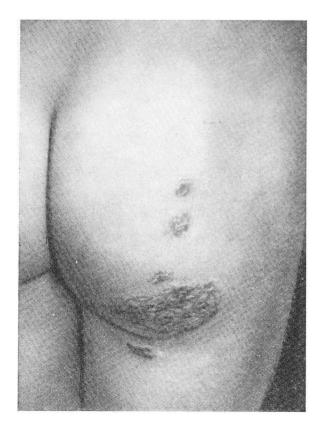
Fig. 29. Superficial trichophytosis of the smooth skin

Trichophytosis of the Nails

The *Trichophyton endothrix* also affects the nails. In the depth of the nail, usually near its free edge, a dirty-grey macule appears and quite rapidly spreads all over the nail. The nail loses its usual lustre, becomes dull and uneven (Fig. 30). The free edge of the nail easily crumbles and therefore appears eaten away.
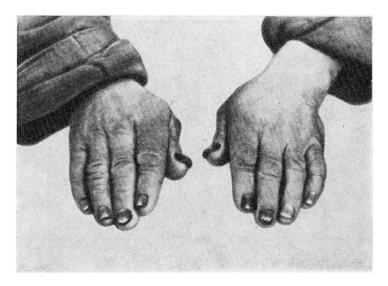
Fig. 30. Trichophytosis of the nails

If the substance of the affected portion of the nail is scraped off with a scalpel, microscopic examination reveals filaments and spores of the fungus.

The nails are infected as the result of extension of the mycotic process from the smooth skin or penetration of the fungus into the nail from the hairy part of the head through scratching. Trichophytosis of the nails is very rarely a separate disease entity. In superficial trichophytosis of the hairy part of the head in children affection of the nails is not so frequent — it occurs in 2 per cent of the patients. One or two finger-nails are usually affected.

Chronic Trichophytosis of Adults

Chronic trichophytosis of adults has a clinical picture and course all its own. This form of the disease affects mainly women, although it is also observed in men. It is produced by the same causative agent—fungi parasitising on the human skin—and begins in childhood as ordinary superficial trichophytosis. But in these

cases it does not pass off by itself during the period of sexual maturation and assumes an unusually protracted, chronic character. The reason for it lies in general organic disturbances. Nervous disturbances and certain endocrine and metabolic disorders are often observed in adult chronic trichophytosis patients. These disorders alter the reactivity of the organism and the clinical picture of the disease. Small, unclearly circumscribed macules with branny scales, short hair fragments and "black dots" are formed on the hairy part of the head. Removal of the scales reveals a pale purplish-reddish skin. In addition to this there are small bald spots with atrophic skin from one to several mm in diameter. Sometimes only atrophic bald spots and but single "black dots" can be found on the head. In such cases a correct diagnosis can be established only by a very careful examination of the entire hairy part of the head and laboratory analysis of the "black dots" and scales for fungi. It is still harder to establish a diagnosis when abundant branny scaling over the entire surface of the head creates the impression of seborrhea. Analysis of the scales for fungi favours a correct diagnosis because hair fragments containing fungi are found in the scales.

In many cases chronic trichophytosis of adults simultaneously involves the hairy part of the head, the smooth skin and the nails.
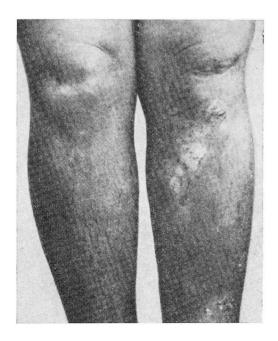
Fig. 31. Chronic trichophytosis of adults
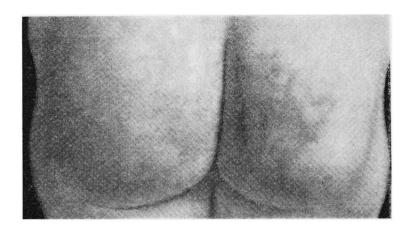
Fig. 32a. Chronic trichophytosis of adults (from A. Arievich
and Z. Stepanishcheva)
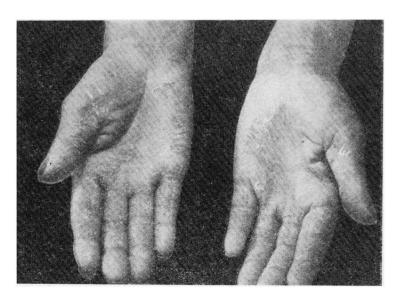
Fig. 32b. Chronic trichophytosis of adults

The lesions in the smooth skin considerably differ in their clinical picture from those of the usual superficial trichophytosis of the smooth skin. In chronic trichophytosis unclearly circumscribed purplish-red, sometimes coalescing exfoliating macules of irregular form and different sizes are formed on the smooth skin. The lesions in the smooth skin are most commonly localised on the knees, shanks, forearms, palms, soles of the feet and the buttocks (Figs. 31 and 32a).

In addition to exfoliation the palms and soles of the feet exhibit hyperkeratosis (Fig. 32b). In some cases patients complain of itching.

Affections of the nails are found in 30 per cent of the cases of chronic trichophytosis of adults, i.e., much more frequently than in children affected with superficial trichophytosis. Usually several or even all finger-nails and often toe-nails are affected.

Chronic trichophytosis of adults is an extremely stubborn disease. Separate little foci on the hairy part of the head and the smooth skin disappear and reappear, but the disease as a whole may, in the absence of treatment, persist all through life.
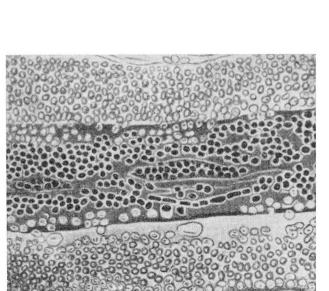
Deep Trichophytosis
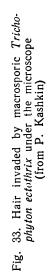
Trichophytons parasitising on animals may also attack man and produce a disease of the hairy part of the head, the beard and mustache, the smooth skin and very rarely the nails.

Microscopic examination reveals that in this form of the disease the fungus not only invades the hair shaft but also arranges itself along its surface—*Trichophyton ectothrix*. For this reason the substance of the hair is destroyed to a much lesser extent and the affected hairs do not break. A sheath of fungus spores arranged in chains is formed along the surface of the hair. A macrosporic *Trichophyton ectothrix* (Fig. 33) and microsporic *Trichophyton* (Fig. 34) are distinguished.

These species of *Trichophyton* are transmitted to man by infected animals. The *Trichophyton ectothrix* evokes a much stronger inflammatory reaction in the skin than does the *Trichophyton endothrix*.

Deep trichophytosis of the hairy part of the head. The disease begins, like superficial trichophytosis, with formation of a pink-red, exfoliating, sharply circumscribed, round macule. However, the picture of the disease quickly changes; the macule develops into an elevated, infiltrated, edematous patch or node on whose surface can be observed numerous follicular pustules, erosions and ulcers at the sites of the ruptured pustules, and purulent crusts. The hairs infected with the fungus fall out because of the suppuration around their follicles (Figs. 35, 36 and 37). The foci
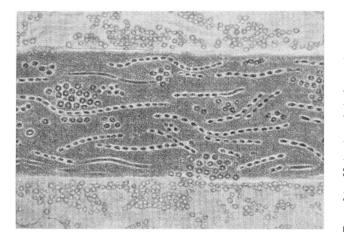
Fig. 34. Hair invaded by microsporic *Trichophyton ectothrix* under the microscope (from P. Kashkin)
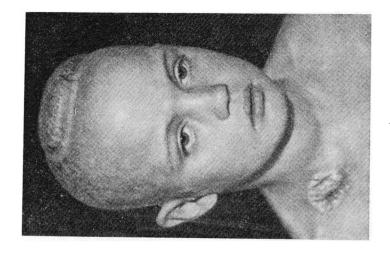
Fig. 33. Hair invaded by macrosporic *Trichophyton ectothrix* under the microscope (from P. Kashkin)

Fig. 36. Deep trichophytosis of the hairy part of the head and of the smooth skin

Fig. 35. Deep trichophytosis of the hairy part of the head

reach the size of a 25-cent coin and grow even larger; they are of a saturated red colour and dense consistency.

Most cases have one or two foci, sometimes more. Upon pressure exerted on the node from the sides the pustules and ulcers discharge drops of pus as though through a sieve (sieve symptom).

Fig. 37. Deep trichophytosis of the hairy part of the face (parasitic sycosis)

Deep trichophytosis shows a tendency to spontaneous healing — the vigorous response reaction of the organism destroys the causative agent. Even in the absence of treatment the inflammatory phenomena subside within a few weeks and cicatrisation begins. The process terminates 3-4 weeks after the onset of the disease with formation of a scar and complete or partial baldness. Proper and timely treatment may considerably reduce this period and diminish the scar and the baldness.

Localised on the head the afore-described picture of deep trichophytosis is known as *kerion celsi*; localised in the beard and mustache it is called *sycosis parasitica*.

Deep trichophytosis of the smooth skin. Few large, round, pink-red foci are formed on the smooth skin. The foci are clearly defined,

infiltrated and elevated above the level of the surrounding skin; on their surface there are follicular pustules, erosions, ulcers and purulent crusts.

A tendency to spontaneous healing is also observed in this form of trichophytosis.

MICROSPOROSIS

This disease is called microsporosis because of the smaller spores formed by the microsporum—its causative agent. In this disease the affected hairs break somewhat higher and the fragments are covered with a sheath of numerous small spores. Under the microscope it can be clearly seen that the spores are outside the hair shafts. Unlike the microsporic *Trichophyton ectothrix* the spores of microsporum are never arranged in chains but assume the form of a mosaic without any definite order (Fig. 38).

Among the causative agents of microsporosis there are also fungi which parasitise on man and animals.

The so-called *Microsporum ferrugineum* affects only man and is very contagious. This form of microsporosis attacks mainly children up to 13 years of age and much less frequently between 13 and 15 years of age. Microsporosis is rarely observed in people past 15 years of age. The *Microsporum ferrugineum* affects the hairy part of the head and the smooth skin. No lesions in the nails are observed.

In microsporosis of the hairy part of the head caused by the *Microsporum ferrugineum* the number of foci and their sizes vary. The foci are often large, of irregular form and unclearly defined. Very often the foci coalesce and form large, irregular figures. The disease is characterised by localisation of the foci at the edge of the hairy part of the head and their partial extension to the smooth skin. In addition to large foci there are usually also many small foci (Fig. 39).

Only some of the hairs break in the foci. The infected hairs break high—3-8 mm above the level of the skin. The hair fragments are surrounded by a sheath of spores. There are usually no inflammatory phenomena, only the periphery of the foci is surrounded by an areola of hyperemia. Hyperkeratosis—a thickening of the stratum corneum in the form of a scale surrounding the hair—is observed at the ostia of the hair follicles.

Microsporosis of the hairy part of the head caused by the *Microsporum canis* is characterised by formation of round sharply circumscribed foci.

This form of the disease usually exhibits one or two large and several small foci.

No inflammatory phenomena are visible; in rare cases the skin is red. All or almost all hair in the foci is broken rather high
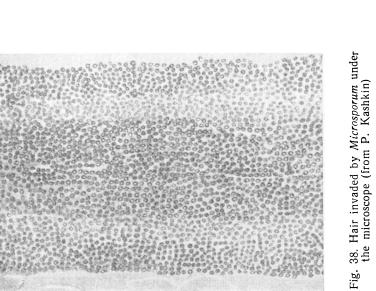
Fig. 39. Microsporosis of the hairy part of the head and of the smooth skin caused by the *Microsporum ferrugineum*

Fig. 38. Hair invaded by *Microsporum* under the microscope (from P. Kashkin)

Fig. 41. Microsporosis of the smooth skin caused by the *Microsporum ferrugineum* (from A. Arievich and Z. Stepanishcheva)
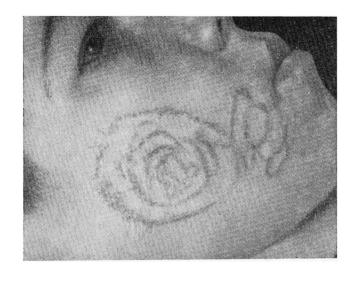
Fig. 40. Microsporosis of the hairy part of the head caused by the *Microsporum canis* (from A. Arievich and Z. Stepanishcheva)

(3-8 mm above the level of the skin) and appears thickened and whitish because of the sheath of spores surrounding the hair shafts. The skin in the foci is all covered with branny scales and appears powdered (Fig. 40).

Exposure of the head of a microsporosis patient to a mercury quartz lamp with a luminescent filtre shows the affected hair to shed a greenish light.

It should be remembered that hair covered with some grease, for example, an ointment or rivanol (2-ethoxy-6,9-diaminoacridine lactate) solution, may appear slightly yellowish-greenish. To avoid an error, it is necessary to examine under the microscope a few hairs which shed a greenish light.

Discovery of the fungus in the affected hair decides the diagnosis.

Microsporosis of the smooth skin closely resembles trichophytosis of the smooth skin. Microsporosis of the skin caused by the *Microsporum canis* is sometimes accompanied by more acute inflammatory phenomena, namely, a bright redness and a peripheral elevation covered with vesicles. Microsporosis caused by the *Microsporum ferrugineum* often exhibits foci in the form of concentric circles, i.e., a new macule appears inside a circle, expands and develops into a ring, then a new macule appears, etc. (Fig. 41).

FÁVUS

The causative agent of favus is the *Trichophyton schoenleini* which parasitises only on man. It invades the hairy part of the head, the smooth skin and the nails, most frequently affecting the hairy part of the head. Some species of this infective agent parasitise on animals and birds and rarely affect man.

Favus of the Hairy Part of the Head

A reddish macule appears on the hairy part of the head and gradually develops into a scutulum which looks like a crust, is round, has a saucer-shaped indentation in the centre and is perforated by a hair. Scutula form about hair follicles and consist of filaments and spores of the *Trichophyton schoenleini* and cells of the stratum corneum of the skin.

The scutula are yellow or the colour of sulfur. In the beginning they are of the size of a pinhead, but gradually expanding grow to several mm in diameter. Larger scutula—up to 1.5 cm in diameter—are sometimes observed. They closely adhere to the skin and are hard to remove. After removal they leave the skin slightly indented, shiny and red. Scutula may coalesce into continuous yellow crusts.

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Detachment of scutula leaves a cicatricial atrophy of the skin. The hair in the affected areas becomes lustreless, grey and dry, appears powedered and lifeless, and resembles the hair of a wig or oakum. Owing to destruction of the hair bulbs the hair at the site of the scutula falls out and never grows again.

Fig. 42. Favus of the hairy part of the head (from A. Arievich and Z. Stepanishcheva)

Favus most commonly begins in childhood, usually from 5 to 14 years of age, although the disease is not infrequently observed in infants. The disease runs an extremely protracted, chronic course. In the absence of treatment it may persist to a very old age. The process terminates in some areas of the hairy part of the head, leaving cicatricial atrophy and baldness, and affects other areas. In the end favus may affect the entire surface of the hairy part of the head except a narrow border along the edge of the hair growth (Fig. 42). The head of a favus patient has a peculiar mousy odour.

Varieties of favus differing from the main scutular form are frequently observed.

In the *impetiginous* form the head is covered, not with scutula, but with crusts which resemble those of impetigo. The *squamous* form of favus is characterised by abundant scales covering the hairy part of the head. However, a correct diagnosis can also be established in these cases since they exhibit grey, lustreless and lifeless hair, and removal of crusts or scales reveals areas of cicatricial atrophy and baldness.

Microscopic examination of the shaft of the hair invaded by the *Trichophyton schoenleini* reveals filaments and spores of the fungus, and air bubbles. The spores and filaments vary in size and shape (Fig. 43). The fungus never fills the entire hair shaft and the hair substance is not destroyed to the extent that it is in trichophytosis and microsporosis. That is why the hair of favus patients does not break, but falls out as a whole owing to atrophy of the hair follicles.

Favus of the Smooth Skin

Favus of the smooth skin usually accompanies favus of the hairy part of the head, but occurs much more rarely than the latter. Scutula (about downy hairs), similar to those forming on the head, or reddish scaling macules simulating a focus of superficial trichophytosis of the smooth skin, are formed on the smooth skin.

Favus of the Nails

Lesions in the nails are also most commonly observed in patients affected with favus of the hairy part of the head and occur in 20 per cent of the adults affected with this dermatomycosis.

The picture of the nail lesions resembles that of trichophytosis of the nails. Yellowish macules appear in the depth of the nail and very slowly enlarging affect the whole nail. The nail grows thick and uneven, and crumbles at the free edge (Fig. 44).

Dermatophytids

Some patients affected with trichophytosis, microsporosis and favus exhibit widespread allergic eruptions—macular, papular and vesicular—on the body and limbs. These lesions do not contain any filaments or spores of fungi. They are called *dermatophytids* (trichophytids, microsporids and favids) and arise as a result of prolonged or strong irritation of the foci of fungus infection. Dermatophytids most commonly appear in patients affected with deep trichophytosis and in cases subjected to excessively irritating local treatment.

EPIDEMIOLOGY OF TRICHOPHYTOSIS, MICROSPOROSIS AND FAVUS

Infection with trichophytosis caused by fungi parasitising on the human skin (*Trichophyton endothrix*) occurs through direct contact of healthy people with patients or through the objects used by patients (clothing, underwear and particularly headgear, combs, hairbrushes, hairclippers, sponges). The disease spreads under conditions of close daily contact, most commonly in the family in
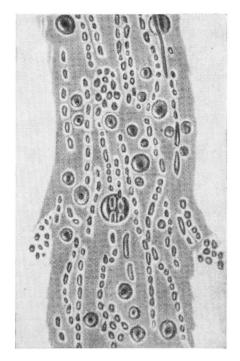
Fig. 43. Hair invaded by *Trichophyton schoenleini* under the microscope (from P. Kashkin)

Fig. 44. Favus of the nails (from A. Zenin and N. Torsuyev)

the absence of adequate sanitation and hygiene. Adults affected with chronic trichophytosis play a particularly important part in spreading trichophytosis among children. A protracted course of this form of the disease is conducive to infecting the people surrounding the patient.

Microsporosis caused by the *Microsporum ferrugineum* is transmitted from patients to healthy people in the same manner as trichophytosis, and is especially contagious. Late diagnosis of this form of microsporosis and inadequate prophylaxis in a children's institution may lead to infection of many children.

Microsporosis caused by the *Microsporum canis* is in most cases transmitted to man by cats infected with microsporosis and less frequently from infected dogs. Children who play with infected cats or kittens easily contract the infection from them. Especially harmful is the habit, prevailing in some families, of allowing children to take a cat to bed with them. An important part in spreading this form of microsporosis is played by stray cats. Infection with this form of microsporosis may also occur through contact of healthy children with patients.

Deep trichophytosis is transmitted to man by infected animals. The macrosporic *Trichophyton ectothrix* parasitises in general on cattle, but less frequently on horses. This variety of trichophytosis affects mainly countryfolk, i.e., persons tending cattle.

The microsporic *Trichophyton ectothrix* parasitises on mice and rats. Gaining entrance into homes these rodents may contaminate the clothing, bedding and other objects by leaving on them their hair invaded by the fungus. Infection with parasitic sycosis may also occur through shaving in a barber shop, if the barber's tools and linens are improperly disinfected.

Favus is less contagious than microsporosis and trichophytosis. It is transmitted through close and long contact, predominantly in the family. The sanitary and hygienic situation in the family is a very important factor. If it is favourable, even the presence of a patient in the family may fail to affect its other members.

Infection with favus is facilitated by certain harmful customs, such as those of continuously wearing some sort of headgear, still prevalent in some communities. Barber shops which violate the rules of fungus disease prevention, especially the rules of disinfection of hairclippers, razors, combs, hairbrushes and linens, may serve as sources of fungus infection of the hairy parts of the head and face.

Prevention of Fungus Infections

The following measures are aimed at preventing fungus infections:

1. *Timely identification, treatment and isolation of patients.* The sooner the fungus-infected patient is discovered and subjected

to treatment the less chance there is for those around to contract the disease. Fungus infection does not cease to be contagious immediately after the beginning of treatment. It is therefore necessary to isolate the patient.

If a fungus disease is contracted by a child in a closed children's institution (children's home, boarding-school, etc.) the child must be hospitalised without fail. Children living in hostels and crowded apartments are also subject to compulsory hospitalisation.

In other cases the measures of isolation depend on the form of the fungus infection. Patients infected with microsporosis caused by the *Microsporum ferrugineum* must necessarily be hospitalised because this disease is particularly contagious. Hospitalisation of adult patients infected with chronic trichophytosis is also desirable because of the stubborn course of this disease and the need for especially thorough treatment. As to other forms of trichophytosis, microsporosis and favus, it is enough to see to it that the patient observes the rules of hygiene at home and carries out the necessary measures of disinfection.

If the patient stays at home, he must continuously wear a linen cap from the moment of establishment of the diagnosis to the end of the treatment. The cap must cover the entire hairy part of the head and closely adhere to it. Its aim is to prevent dissemination of scales and falling-out or broken fungus-infected hair.

To prevent dissemination of fungus-containing hair fragments and scales by the patient, *glue caps* have also been proposed (see Supplement, p. 334.). The patient's regimen during the treatment must prevent the spread of fungus infection. A special place is provided for the storage of the patient's outer garments and headgear. The patient must have a separate bed, separate towel and separate sponge for washing. The dirty underwear must be kept in a special bag or pillowcase. A fungus-infected patient must not be allowed close contact with healthy children, to play with them, etc. Children must not be admitted to children's institutions (nurseries, kindergartens, schools, etc.) until completely cured.

2. *Disinfection of linens, clothing and other objects.* Hair fragments and scales containing fungi may long be found on the linens, clothing, combs and other objects used by the patient. In the hair fragments the fungi may retain their virulence for several months and even years. All objects contaminated by hair and scales must therefore be disinfected. The clothing and linens are disinfected in formalin-vapour or air-steam chambers. Simpler methods of disinfection may also be used, namely, soaking the linens for one hour before washing in a 1 per cent chloramine solution, boiling them for 15 minutes, and pressing the clothing with a hot iron through a moist cloth. In the absence of a disinfection chamber the outer garments may be exposed to the sun for 2-3 weeks in summer time,

and thoroughly cleaned with snow and then pressed with a hot iron in winter. Headgear is very difficult to disinfect and should therefore be burned.

Current and *final disinfections* are distinguished. Current disinfection is carried out in the course of the patient's treatment. It is aimed at systematically decontaminating the objects which are used by the patient and may therefore serve as the source of infecting the people surrounding the patient.

All of the utilised dressing material, fallen-out hair and cutoff or removed nails must be burned without fail.

The room occupied by the patient must be cleaned daily with moist rags and the rags must be disinfected by boiling for 15 minutes.

The final disinfection is carried out at the end of the treatment.

The patient's clothing and linens are disinfected in a disinfection chamber. The best method is disinfection in a formalin-vapour chamber for 2.5 hours, using 250 ml of formalin per 1 m³ at a temperature of 58-59°C. Simple steam disinfection at a temperature of 110-111°C and a pressure of 0.5 atm for 1.5 hours may also be employed. While the clothing and linens are being disinfected the floor in the patient's room is flooded for 1 hour with a 5 per cent chloramine solution and then washed with warm water and soap. On the same day all members of the family wash and change their linens.

3. *Determination of the sources of infection and examination of persons who had contact with the patient.* In each case of trichophytosis, microsporosis and favus it is very important to determine the source of the patient's infection. In cases of superficial trichophytosis of the hairy part of the head or the smooth skin the source of infection must be sought primarily in the patient's family, among the adults. This applies particularly to favus patients because infection with favus always occurs under conditions of close daily contact in an unsatisfactory hygienic situation. In cases of trichophytosis and favus special attention must be devoted to examining the members of the patient's family living together with the patient. The adult members of the family must be examined at least 3 times: upon discovery of the child's infection, during the child's treatment and after the cure. In cases of microsporosis caused by the *Microsporum ferrugineum* the source of infection must be sought among the children with whom the patient had contact. In cases of microsporosis caused by the *Microsporum canis* a veterinarian must examine the cats and dogs with which the patient had contact. Irradiation of the examined dogs by means of a mercury vapour lamp with a luminescent filtre in a dark room greatly facilitates the diagnosis of microsporosis. In cases of deep trichophytosis caused by the macrosporic *Trichophyton ectothrix* the veterinarian must examine the cattle with which the patient had contact.

In cases of deep trichophytosis caused by the microsporic *Trichophyton ectothrix* the sanitary and epidemiological services must exterminate the rats and mice in the patient's dwelling because these rodents are the source of human infection with this form of trichophytosis.

It follows from the above that fungus infections cannot be successfully prevented without knowledge of their clinical picture and epidemiology.

In all cases of trichophytosis, microsporosis and favus it is necessary to examine all those who had any contact with the patient — members of his family, his playmates at home and in the children's group. Among them there may be some who contracted the infection from the same source or from the given patient. A single examination is not enough. In these diseases the incubation period may be 2-3 weeks and in cases of infection with the *Microsporum ferrugineum* even 6 weeks. That is why examinations are conducted every 5-7 days over the aforementioned periods of time.

4. *Prevention in children's institutions.* The following measures are carried out in children's institutions:

(a) examination of all children newly admitted to the institution or children returning after illness, departure, quarantine, vacations, etc.;

(b) regular monthly examinations of all children;

(c) examination of all persons hired for work in children's institutions and their periodic, monthly examinations. The prophylactic examinations of children and adults must include a thorough examination of the hairy part of the head, the skin and the nails;

(d) isolation of all persons suspected of fungus infection and their immediate hospitalisation for the entire period of treatment upon discovery of a fungus infection;

(e) prohibition of cat and dog pets in children's institutions in areas where there are cases of microsporosis caused by the *Microsporum canis*;

(f) cutting of the hair of all children in children's institutions is desirable. Before cutting their hair all children must be examined. The hair of children with a normal scalp is cut first. After each haircut the hairclippers are immersed in alcohol and held over the flame of a burner. Children with phenomena of scaling, redness or crusts on the head are the last to be given haircuts. After cutting the hair of each such child the hairclippers are disassembled, the parts are cleansed of the hair, immersed in alcohol and held over the flame of a burner;

(g) upon discovery of a fungus infection in a children's group all children are examined once a week for 2-3 weeks. In cases of microsporosis caused by the *Microsporum ferrugineum* the examinations are conducted every 4-5 days for a period of 6 weeks. In

closed children's institutions a quarantine is declared for the same period of time.

The usual rules of hygiene must be strictly observed in all children's institutions. The children must have individual combs. caps and towels.

5. *Prevention of fungus infections in barber shops.* Prevention of fungus infections in barber shops is based on strict observation of sanitary rules, obligatory disinfection of linens and tools, and regular medical examinations of the workers of barber shops. Change of sheets, napkins and other linens for each client, use only of sterilised shaving brushes, hand washing by barbers before attending to each client, and thorough cleaning of the premises are obligatory. The used linens are subject to disinfection. The sheared hair must be collected and immediately burned. The brushes and combs are cleansed of the hair by means of cotton and disinfected in a 5 per cent chloramine solution.

Workers of barber shops must not cut the hair of persons with diseases of the hairy parts of the head and face without permission of a physician.

Washing of the head with soap and hot water after a haircut is also a good measure for preventing fungus infection. The head must be washed immediately upon coming home from the barber shop.

TREATMENT OF TRICHOPHYTOSIS, MICROSPOROSIS AND FAVUS

Epilation (removal of the hair)ˇis a sine qua non of successful treatment of trichophytosis, microsporosis and favus. Epilation favours deeper penetration of antiparasitic agents into the hair follicles.

The principal method of epilation is roentgen irradiation of the hairy part of the head.

After roentgen irradiation the hair is painlessly removed by means of forceps or even falls out itself. In mechanical, manual epilation the hair is removed with difficulty, the epilation is painful and the fungus-infected hairs often break. Roentgen rays not only cause epilation, but also produce a therapeutic effect by creating unfavourable conditions for the development of the fungus in the skin.

Before irradiation the head is cleansed of crusts and scutula by means of an oil compress and the hair is cut 1-2 cm above the level of the skin. If the skin on the hairy part of the head exhibits inflammatory phenomena or secondary pyogenic infection, the initial treatment is aimed at eliminating them. No application of medicinal substances to the head must precede or follow irradiation until the hair has fallen out because of the danger of irritating the skin.

Careless and unskillful roentgen irradiation may produce roent-
gen dermatitis, ulcers, atrophy of the skin and permanent baldness.

For roentgen irradiation the head is divided into 4, less fre-
quently 5, fields which are irradiated alternately, one field a day.
Each field is given an epilation dose of roentgen rays in one ex-
posure.

Children 3-5 years of age are given a divided roentgen epilation
dose, i.e., the epilation dose of roentgen rays is divided into
2-4 parts and each field is irradiated 2-4 times at intervals 2-4 days.
Roentgen epilation for children under 3 years of age is contrain-
dicated.

Daily washing of the head with warm water and soap is pre-
scribed 11-12 days after roentgen irradiation.

This facilitates the falling-out of hair, which begins 11-12 days
after irradiation. The hair usually ceases to fall out between the
18th and 20th days after irradiation, at which time removal of
the remaining hair and its fragments is begun and antiparasitic treat-
ment is instituted. All hair fragments and the remaining hair are
removed with forceps, and the "black dots"—with a syringe
needle. The hairs are grasped with epilation forceps, one at a time,
and is extracted in the direction of their growth. With this technique
the affected hair breaks less frequently. The foci of infection and
a zone of 1-2 cm around them are epilated with particular care. The
epilation must not take more than 4-5 days. Wilkinson's ointment
is rubbed into the skin of the head every night and the head is
washed with warm water, soap and a sponge in the morning. After
washing the head is painted with a 2 per cent iodine tincture. In
favus, chronic trichophytosis and microsporosis, caused by the
Microsporum ferrugineum, such treatment is administered for a
period of 6-8 weeks, and in other forms of fungus infection of the
hairy part of the head—for 4-5 weeks.

Good results are produced by the "exfoliation" method
proposed by A. Arievich. The method consists in the following.
From 2 to 3 days after the hair has fallen out an ointment consist-
ing of 12 per cent salicylic acid and 6 per cent lactic (or 3 per cent
benzoic) acid in vaseline is applied to the entire head. In cases of
children up to 5-6 years of age the same ointment must be applied
in half the above concentration. After application of the ointment
the entire head is covered with wax-paper and bandaged. The
bandage is left on for 48 hours. This is followed by application of
a 3 per cent salicylic ointment dressing for 24 hours. Thereafter
the loosened stratum corneum is easily removed by means of a
cotton tampon or a dull scalpel. The remaining hair and its frag-
ments in the foci of affection and in the zone of 1-2 cm around them
are removed with epilation forceps. During the following 10 days
the head is washed daily with warm water and soap, and a sponge
or brush. Then the "exfoliation" is repeated and the head is

washed every day again for 2-3 weeks. If hair fragments and black dots persist after the second exfoliation, a third exfoliation is performed. In cases of chronic trichophytosis of adults, microsporosis caused by the *Microsporum ferrugineum*, and multiple foci of superficial trichophytosis 3 exfoliations are recommended. In the intervals between the exfoliations Wilkinson's ointment is rubbed in every night and the head is washed in the morning, after which it is painted with a 2-5 per cent iodine tincture.

Vigorous treatment after roentgen epilation sometimes produces dermatitis of the hairy part of the head—redness, edema, exudation, itching, burning and pain. In such cases the antiparasitic treatment is suspended until cessation of the acute inflammatory phenomena, and treatment, as for dermatitis, is prescribed.

After roentgen irradiation it takes 2-3 months for the hair to grow back all over the head. To check on the cure of the fungus infection, the hair and scales are examined for fungi in the laboratory 3-4 days after the end of the treatment. If the results are negative, the examination is repeated in 3-4 days. Twofold negative results of laboratory examination warrant the return of the child to the children's group provided the total treatment was adequate (at least 6 weeks after the hair fell out in cases of favus and microsporosis caused by the *Microsporum ferrugineum*, and at least 4 weeks in all other cases of fungus infection of the hairy part of the head). However, the patients must be kept under medical observation until the hair has completely grown back all over the head.

If the fungus infection has recurred after roentgen irradiation, treatment with roentgen rays may be repeated no sooner than within 6 months.

If roentgen therapy is contraindicated, fungus infections are treated without roentgen rays. The other methods are less reliable than roentgen therapy, but they often effect a cure and in all cases prevent the spread of the mycotic process and the infection of the people surrounding the patient. That is why such treatment must be administered to all patients who cannot be given roentgen therapy.

One of the healing agents, when no roentgen rays are used, is a 3 or 5 per cent thallium plaster, if the foci of affection, are few (see Supplement, p. 334.).

If the foci of affection are too numerous or very large, a thallium plaster cannot be used.

In such cases manual epilation with forceps is performed in combination with Arievich's exfoliation method (once in 10 days), and antiparasitic treatment is administered. The treatment in these cases must continue at least 3-4 months.

In recent years Soviet scientists (A. Arievich and V. Zasosov et al) have developed a new preparation for treating fungus infec-

tions of the hairy part of the head—epilin—and have elaborated a method of its application. Application of a 4 per cent epilin plaster to any part of the scalp for an average of 20 days results in falling-out of all the hair on the head, this epilation hardly differing qualitatively from roentgen epilation.

The new method of general treatment based on peroral administration of the antimycotic antibiotic griseofulvin (fulcin, fulgin).

The antibiotic is dispensed in 250 mg pills. The dose of griseofulvin depends on the patient's age. Children up to 3 years of age are given 125 mg (half a pill) twice a day, children from 4 to 7—250 mg twice a day, from 8 to 16—250 mg 3 times a day, and adults—250 mg 4 times a day. For the treatment of patients with fungus infection of the hairy part of the head griseofulvin is usually administered daily during the first week and then in the same doses on alternate days for another 3-5 weeks.

External treatment is administered simultaneously with griseofulvin therapy, i.e., the hair is shaved off once a week and the head is washed, painted with a 2 per cent iodine tincture and covered with a sulfur-pitch ointment (10 per cent sulfur and pitch each in vaseline) every day. Arievich's exfoliation method is useful in cases of multiple foci of affection on the head. If there are lesions in the nails, removal of the affected nails and application of antiparasitic agents are recommended. Griseofulvin is usually well tolerated by patients, but sometimes it produces side effects, namely, toxicoderma, thirst, headache, nausea and abdominal pain. The patients therefore require careful watching during the treatment. The experience of griseofulvin treatment attests its effectiveness in trichophytosis, microsporosis, and favus of the hairy part of the head and of the nails.

The *treatment of deep trichophytosis* of the scalp and beard consists in removing the hair in the foci and in a zone of 1 cm around them with epilation forceps and application of anti-inflammatory and disinfecting lotions and moist-desiccant dressings of a 1 : 1,000 rivanol solution and a 5-10 per cent ichthyol solution. As the acute inflammatory phenomena subside, dressings with disinfecting ointments (5 per cent xeroform, 2-3 per cent ammoniated mercury, sulfur-pitch, etc.) are applied.

Treatment of lesions in the smooth skin. The foci on the smooth skin are painted once or twice a day with a 5 per cent iodine tincture or are inuncted with Wilkinson's ointment or a 10-20 per cent sulfur ointment. The lesions in the smooth skin require for the most part 7-10 days to heal.

In cases of more stubborn lesions in the smooth skin due to infection of the downy hair with the fungus the foci are painted with a 10 per cent salicylic acid solution, 10 per cent lactic acid and 5 per cent resorcinol in collodion (A. Arievich).

The foci are painted 4-5 days in succession and then a dressing with 2-5 per cent salicylic ointment is applied. After this the film of collodion is easily removed together with the downy hair. This treatment is repeated 2-3 times.

The first to be used *for the treatment of deep trichophytosis of the smooth skin* are moist-desiccant dressings with a 5-10 per cent ichthyol solution; after abatement of the acute inflammatory phenomena the foci are painted with a 2 per cent iodine tincture and are inuncted with Wilkinson's ointment.

The *treatment of the lesions in the smooth skin in cases of chronic trichophytosis of adults* is much more complicated. In these cases Arievich's exfoliation method is used once in 10 days, antiparasitic agents are applied, the foci are pasted up with strips of adhesive plaster, and general treatment is administered.

The *treatment of onychomycoses* (fungus infection of the nails) is based on removal of the infected nails and subsequent application of antiparasitic agents for the treatment of the nail bed. The most favourable results in the treatment of onychomycoses are produced by means of *keratolytic* and *fungicidal* plasters proposed by A. Arievich and B. Lebedev. Onycholysin proposed by G. Andriasyan is a good agent and A. Araviisky's method is very helpful in removing the nails (see Supplement, p. 335).

Nail lesions are one of the most stubborn manifestations of trichophytosis and favus and are the most difficult to treat. Their treatment is not infrequently followed by relapses.

General treatment of patients with fungus infections. Many cases of trichophytosis, favus and microsporosis cannot be limited merely to topical, external treatment of the foci of affection because the general disturbances in the organism are conducive to a stubborn course of the fungus infection and relapses after the treatment.

Favus patients often exhibit anemia, undernourishment and general debility. Fish liver oil, preparations or iron and arsenic, vitamins C, A and B_{12}, and campolon (aqueous liver extract) are very helpful in these cases. The same applies to patients with superficial trichophytosis and microsporosis in whom similar phenomena are observed.

General treatment is particularly important in cases of adults infected with chronic trichophytosis. In addition to general roborant treatment these patients are prescribed, if indicated, endocrine preparations—diethylstilbestrol, folliculin, sinestrin (diethylstilbestrol dipropionate) for women and methyltestosterone for men. In cases of lesions in the smooth skin and the nails a good effect is always produced by a circular novocain block which is repeated 4-6 times at intervals of 4-6 days. *Griseofulvin* is very helpful in the treatment of chronic trichophytosis of adults.

Control of Fungus Infections in the U.S.S.R.

The importance of trichophytosis, microsporosis and favus is determined by the contagiousness of these diseases, their ability to affect masses of people and their stubborn, chronic course. The spread of fungus infections depends on social and hygienic conditions. In some countries fungus infections often become extremely widespread.

The Soviet public health services have devoted a great deal of attention to the control of fungus infections. The patients have been provided with free and qualified medical aid. Special expeditions were sent to some of the national districts in order to eradicate the mass fungus infections. The expeditions examined the population and treated the patients they discovered. A network of mycological hospitals and dispensaries and roentgen offices was established for the treatment of fungus-infected patients, and accurate records of these patients were kept. As a result of the work done, the incidence of dermatomycoses of the U.S.S.R. has considerably diminished. The experience of the Soviet public health services shows that successful control of dermatomycoses requires:

1. Accurate records of fungus-infected patients.

2. Employment of dispensary methods—(a) dispensary observation of every patient until cured, (b) ascertainment of the source of infection and adoption of corresponding measures (treatment of the patient, enlistment of the services of veterinarians for treating or exterminating infected animals, deratisation, etc.), and (c) examination of the members of the patient's family and other persons who had contact with the patient.

3. Regular mass prophylactic examinations of children in schools and other children's institutions, and of children who are not members of children's groups.

4. Health education, i.e., dissemination among the population of correct information concerning the manifestations of fungus infections, the ways of their transmission and measures for their prevention.

The success of these measures largely depends on the efficient performance by intermediate medical workers of their duties.

EPIDERMOPHYTOSIS

Epidermophytosis is caused by fungi of the genus *Epidermophyton*. These fungi parasitise only on man; epidermophytosis most commonly occurs on the feet.

Epidermophytosis usually affects the skin of the soles of the feet and the interdigital folds, especially those between the 4th and 5th and between the 3rd and 4th toes. The onset and develop-

ment of epidermophytosis are favoured by hyperidrosis and uncleanliness of the feet, intertrigo, excoriations and flatfoot. Infection with epidermophytosis occurs under conditions of close family contact (common bed, common shoes, socks and stockings) and in various bathing establishments (showers, baths), especially if these are maintained in unsanitary conditions. Epidermophytosis patients cast off scales of the skin thereby disseminating the fungi and spreading the disease. In the scales of the skin, in shoes, socks and stockings, on the floors, wooden gratings and mats in showers and baths the *Epidermophytons* long remain viable and pathogenic.

According to the clinical picture, squamous, intertriginous and dyshidrosiform epidermophytoses are distinguished.

Squamous epidermophytosis manifests itself as lamellar scaling of the skin of the soles and interdigital folds of the feet, mainly between the 4th and 5th and the 3rd and 4th toes. Microscopic examination of the scales reveals filaments of the *Epidermophyton*. Squamous epidermophytosis does not exhibit any appreciable inflammatory phenomena and usually produces no subjective sensations. Only now and then superficial fissures between the toes are formed and a slight itching is observed.

The intertriginous form is characterised by maceration of the epidermis in the interdigital folds and intense itching. The skin in the interdigital folds becomes whitish and rugose, the epidermis exfoliates in layers, revealing a pink-red or shiny saturated-red surface.

The macerated epidermis often desquamates leaving only an overhanging border along the edges of the affected area. Fissures and erosions with a copious serous exudate may arise in the depth of the interdigital fold and on the contacting surfaces of the toes.

In dyshidrosiform epidermophytosis intensely itching vesicles varying in size from that of a millet seed to that of a bean appear in the interdigital folds, on the lateral surfaces of the toes and on the soles of the feet (Fig. 45) in the region of the arch. The vesicles sometimes lie deep in the skin and are seen through the epidermis as sago grains. They may open and develop into painful erosions. The process not infrequently assumes the character of eczema, which is due to the ability of the *Epidermophyton* and its waste products to cause allergic states in the organism.

Epidermophytosis is quite often complicated by secondary pyoderma and, instead of the vesicles, pustules surrounded by a red areola, erosions, ulcers and crusts are formed. Edema and sharp pain, which often makes walking difficult, appear. Sometimes lymphangitides develop.

The allergic state caused by the *Epidermophyton* and its waste products may lead to eczematous phenomena on the arches of the feet and reflex development of dyshidrosiform vesicles on the palms, on the fingers and other portions of the skin. The dyshidrosiform
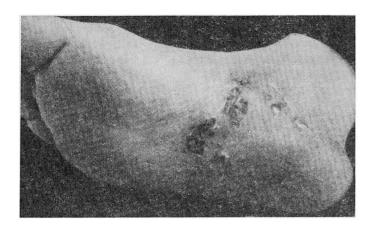
Fig. 45. Dyshidrosiform epidermophytosis (from A. Arievich and
Z. Stepanishcheva)

and eczematous foci on the palms of the hands and in other areas,
as well as the macular and papular eruptions in patients with epi-
dermophytosis of the feet are called epidermophytids. In epider-
mophytosis patients the toe-nails, most commonly the nails of
the large toes, may also be affected with the fungus. Yellow lines
and round or irregularly-shaped macules appear closer to their
free edge, in the depth of the nails; the nails thicken and then
crumble.

Treatment. Methods of general treatment are indicated for
patients with dyshidrosiform epidermophytosis, with epidermo-
phytids and sometimes with intertriginous epidermophytosis. In
such cases calcium chloride, sodium bromide, novocain and vita-
min B_1 are administered intravenously or per os.

External topical treatment of epidermophytosis plays an im-
portant part and depends on the form of epidermophytosis.

Daily warm baths with potassium permanganate for the feet
are prescribed for dyshidrosiform and sometimes intertriginous
epidermophytosis. After the bath the vesicles are cut off with
scissors and moist-desiccant dressings with rivanol (1 : 1,000) are
applied.

As the phenomena of acute inflammation abate, pastes and
ointments with naphthalan oil (10-50 per cent), sulfur (2-5 per
cent) and pitch (2-5 per cent) are applied.

The same baths and applications of a disinfecting (fungicidal)
solution, according to Suteyev and Asnin's prescription, undecyne
ointment or nitrofungin are prescribed for intertriginous epider-
mophytosis (see Supplement, p.326—327). The usual 1 per cent
methyl violet or brilliant green solution in 140 proof alcohol may
also be used.

In the squamous form of the disease the foci of affection are painted with the same solutions or with a 1-2 per cent iodine tincture. Baths with potassium permanganate to be followed by removal of the scales with a forceps or scissors are prescribed once every 2-3 days.

To prevent relapses after disappearance of the phenomena of epidermophytosis and termination of the treatment it is necessary to keep the feet absolutely clean. It does well to dust the soles and interdigital folds of the feet with a powder composed of tannin, zinc oxide, sulfur, salicylic acid and talcum (see Supplement, p.327).

Prevention of relapses of epidermophytosis also requires disinfection of the patient's socks, stockings and shoes. A piece of cotton soaked in a 40 per cent formalin solution is placed in the shoes for the night. Another method of disinfecting the shoes consists in wiping the insoles and inner surfaces of shoes with a 10 per cent formalin solution and in placing a piece of cotton soaked in a 10 per cent formalin solution in the shoes for the night. The patient's linens, stockings, socks and drawers must be boiled before washing. Individual prevention of epidermophytosis consists in treatment of hyperidrosis of the feet, intertrigo and excoriations, elimination of tight-fitting shoes and keeping the feet absolutely clean.

To avoid infection with epidermophytosis one must not use anybody else's socks, stockings, shoes, linens, bed, and sponge. One must not walk barefooted on floors of rooms occupied by several persons. In cases of hyperidrosis powdering with urotropin is prescribed.

On the first day the well-washed and towelled soles and interdigital folds of the feet are powdered with urotropin, and the patient puts on clean socks or stockings. The layer of powder is renewed daily over a period of 6-7 days, during which time the patient neither washes his feet nor changes his socks or stockings.

Patients with flatfeet are prescribed orthopedic shoes.

Public measures for preventing epidermophytosis consist in maintaining strict cleanliness in showers, baths and swimming pools, and in disinfecting their mats, floors and wooden gratings.

Pityriasis Versicolor and Erythrasma

Pityriasis versicolor and erythrasma are fungus skin infections characterised by an absence of inflammatory phenomena and are but barely contagious. Even long and close contact between a healthy person and a patient (for example, man and wife) usually fails to produce the disease and the infection is transmitted only in cases of predisposition associated mainly with excessive sweating. In both these diseases the fungus parasitises only in the stratum corneum and causes no response inflammatory reaction in

the deeper layers of the skin. Only some cases are accompanied by a mild itching and slight redness of the skin.

The fungus causing pityriasis versicolor produces yellowish, reddish and brown macules on the chest, back, neck and, less frequently, the extremities. The macules are covered with branny scales which increase in number on scraping.

Erythrasma is localised mainly on the medial surfaces of the thighs (in men at the points of contact with the scrotum), in the axillae and under the breasts. The eruption of erythrasma consists of red or red-brown macules. The macules often coalesce and form large scalloped foci. The surface of the macules is covered with branny scales. On scraping their number increases. The disease is but rarely accompanied by a mild itching.

The macules of pityriasis versicolor and erythrasma occur as the result of accumulation of filaments and spores of the fungus and of loosening of the stratum corneum.

Both diseases run a chronic course, may persist for many years and recur after an apparent cure.

The *treatment* of these dermatomycoses consists in application of fungicides and agents which exfoliate the stratum corneum. Inunctions of a 10-20 per cent sulfur ointment, rubbing-down of the affected portions of the skin with 4-5 per cent salicylic alcohol, and Demianovich's method (see Treatment of Scabies, pp. 138-139) are used in the treatment of pityriasis versicolor. The same agents are used in the treatment of erythrasma, but in smaller concentrations in view of the possibility of irritating the more sensitive and delicate skin in the area of large folds (5-10 per cent sulfur ointment, 2 per cent iodine tincture, etc.).

Strict bodily cleanliness, frequent washing and changing of underwear, rub-downs with 1-2 per cent salicylic alcohol, and sun baths are recommended for prophylactic purposes.

YEAST INFECTIONS OF THE SKIN AND MUCOSA

Yeast and yeastlike fungi are widely distributed in our surroundings. The *Candida*, a genus of yeastlike pathogenic microorganisms, plays the most important part in human pathology. The diseases caused by these microorganisms are called candidids. The microorganisms of the genus *Candida* are always found as saprophytes on various fruits and vegetables, as well as on the skin and mucous membranes of man and animals. Under certain conditions which reduce the resistance of the organism these microorganisms become virulent and may cause various diseases in man. Such conditions present themselves in emaciated people, in diabetics, and in patients affected with severe toxemias of pregnancy and other diseases. Prolonged use of antibiotics plays a particular role in the development of candidids. Antibiotics,
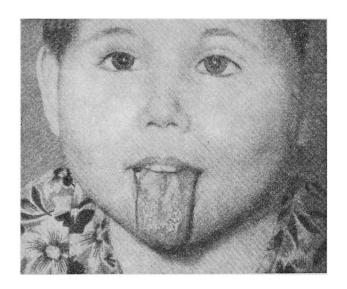
Fig. 46a. Candidid (thrush)
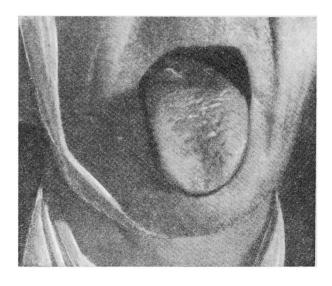
Fig. 46b. Candidid. Glossitis developed in pyoderma
patient treated with tetracycline
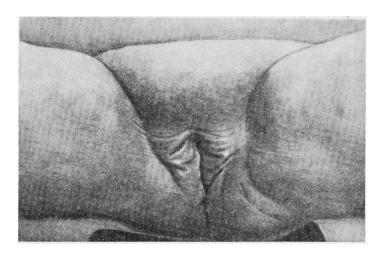
Fig. 47. Candidid of the genitalia and inguinal folds in a diabetic patient

Fig. 48. Candidid under the breasts
(from P. Kashkin)
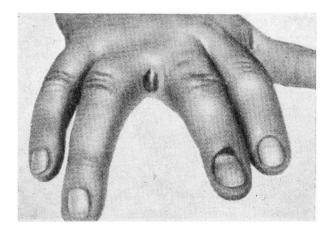
Fig. 49. Yeast erosion between the fingers (from A. Arievich and Z. Stepanishcheva)
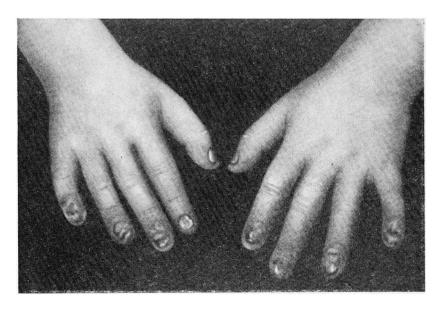
Fig. 50. Candidid of the nail folds and nails

especially those with a broad spectrum of antibacterial action, such as biomycin and synthomycin, depress not only the causative agent of the current disease, but also cocci, bacilli and other microbes—normal saprophytes of the skin and mucous membranes sensitive to antibiotics. At the same time the saprophytes which are not sensitive to antibiotics, as for example, the fungi of the genus *Candida*, begin to multiply vigorously and to acquire pathogenic properties.

Thrush—one of the most frequent manifestations of candidid—most commonly occurs in infants, usually during the first days and weeks of life. It is ordinarily observed in debilitated children suffering from dyspepsia or other children's diseases.

Whitish spots the size of a millet seed or a lentile appear on the mucous membranes of the tongue, cheeks and palate (Fig. 46 a). Sometimes the spots coalesce and become continuous. Upon disappearance the spots leave a bright-red, sometimes bleeding, mucosa. The infant becomes restless, sucks poorly and loses weight. In emaciated children this disease may run a severe course, affect the mucosa of the fauces, esophagus, intestines and lungs, and may end lethally.

In older children and in adults thrush is observed mainly in severe diseases; in debilitated patients it occurs as the result of prolonged use of antibiotics.

There are also other clinical forms of yeast affections of the mucous membranes (Fig. 46b). Affection of the corners of the mouth, so-called yeast perlèche, and affection of the vaginal mucosa (vaginal moniliasis) are relatively common occurrences.

The fungi of the genus *Candida* cause numerous and various skin affections. Lesions in the skin folds—inguinal (Fig. 47), anal region, under the breasts (Fig. 48), and interdigital folds on the hands and feet (Fig. 49)—are most commonly observed. The candidids of the skin folds are most frequently superficial bright-red erosions surrounded by an areola of macerated whitish stratum corneum. The borders of the lesions are sharply and irregularly outlined, often scalloped. Patients complain of itching and burning.

The fungi of the genus *Candida* very often affect the nail folds and nails (most commonly of the fingers). The nail folds, especially the posterior one, thicken, become purplish-red, painful, edematous and compact, and the nail folds disappear. The nails become uneven, lustreless, blackish-brown in the region of the lunulas and side edges, and sometimes detached from the nail beds (Fig. 50).

The fungi of the genus *Candida* may also cause various lesions in the internal organs, to the point of yeast sepsis (generalised candidid).

Treatment. Measures aimed at improving the patient's general condition and treatment of concurrent diseases are of the utmost

importance. An appropriate regimen is established and the patient is prescribed a vitamin-rich diet. Antibiotics are contraindicated or must be administered with extreme caution. The most important agent for general treatment is the antibiotic nistatin which is administered to adults per os in a dose of 500,000-1,000,000 u 3-4 and more times per day, depending on the severity of the disease. The daily dose of nistatin is 2,000,000-5,000,000 u and in severe generalised candidid—up to 10,000,000 u. Administration of the B complex vitamins—thiamine, riboflavin and nicotinic acid, of vitamins K and C, and potassium iodide in increasing doses (2-10 per cent solution in tablespoonfuls) is useful. Applications of 1-2 per cent aniline dyes—methyl violet, gentian violet and crystal violet—are used for topical treatment. Solutions in 140 proof alcohol are used for application to foci of affection on the skin, and water solutions of the dyes for the mucous membranes. Good effects are produced by applications of a 10 per cent solution of borax in glycerin, 5-10 per cent silver nitrate solution, 2-10 per cent blue vitriol solution, and for gargling—a 2-5 per cent soda or boric acid solution.

Excellent effects are produced in many cases by nistatin ointment containing 100,000 u of nistatin per 1 g of ointment base.

Prophylaxis. It is necessary to watch the general condition and the state of the skin and mucous membranes of the patients receiving antibiotics. In doubtful cases specimens are obtained by scraping the oral and vaginal mucosa and the foci on the skin; the specimens are microscopically examined in a 15-20 per cent caustic lye for fungi of the genus *Candida*.

During antibiotic treatment it is recommended to administer to patients thiamine, riboflavin, nicotinic acid, vitamin K and ascorbic acid prophylactically.

TUBERCULOSIS OF THE SKIN

Tuberculosis is one of the most widespread infectious diseases. The social conditions of capitalist society are conducive to wide-spread tuberculosis.

Tuberculosis is a very serious infection which causes deep changes in the patient's organism.

Tuberculosis of the skin is one of the manifestations of the tuberculous infection in man. It must not be considered a local disease since it involves general changes in the organism of the patient. Many patients suffering from tuberculosis of the skin (25-32 per cent) are found to have active pulmonary tuberculosis. In most cases of tuberculosis of the skin and lungs the pulmonary process runs a benign course.

According to O. Podvysotskaya, 30 per cent of lupus patients simultaneously have a tuberculous affection of the bones, mainly the small bones of the feet and hands. Tuberculosis of the bones may sometimes be discovered only roentgenologically. Positive skin reactions to tuberculin (Pirquet test, etc.) are a sign of altered reactivity of the organism of patients affected with tuberculosis of the skin.

The causative agent of tuberculosis—*Mycobacterium tuberculosis* (tubercle bacillus—abbreviated, t.b.)—gains entrance into the skin either from the internal organs affected with tuberculosis or from the exterior. From the external environment the tubercle bacillus enters the skin comparatively rarely. In some patients lupus developed on the earflap after a puncture for wearing earrings, if the puncture was made by a person with active pulmonary or laryngeal tuberculosis, the needle used for the puncture being moistened by his saliva.

There have been cases of tuberculosis of the skin contracted by workers of dissecting rooms or morgues, as the result of wounds sustained during post-mortem examinations of bodies of people who had had active tuberculosis while alive, and by workers of slaughterhouses and meat-packing plants while dressing the carcasses of animals infected with tuberculosis.

In the overwhelming majority of cases the causative agent of tuberculosis gains entrance into the skin from the internal organs infected with tuberculosis, most commonly from the lungs, lymph nodes and bones. Hence, tuberculosis of the skin is almost always a secondary manifestation of tuberculous infection of the organism.

Primary tuberculosis of the skin, i.e., development of tuberculosis of the skin in man formerly uninfected with tuberculosis, is a very rare occurrence.

From the organs affected with tuberculosis the tubercle bacilli may penetrate into the skin in various ways.

1. *Lymphogenic way.* This way the causative agent of tuberculosis penetrates through lymph spaces and vessels directly into the skin near the focus of affection. It is believed that tubercle bacilli often gain entrance into the skin in this manner in scrofuloderma. The frequent localisation of lupus on the face is due to penetration of tubercle bacilli into the skin through the lymphatics from the cervical and submaxillary lymph nodes infected with tuberculosis. The tuberculous process in a lymph node renders the flow of lymph difficult, which in some areas makes the lymph flow back from the lymph node to the tissue lymph spaces in the skin. In such cases the lymph flow carries the tubercle bacilli.

2. *Hematogenic way.* Destruction of blood vessels in tuberculous foci of internal organs enables the causative agent of tuberculosis to gain entrance directly into the blood stream. Tubercle bacilli may also invade the circulatory system through lymph vessels. Having entered the blood tubercle bacilli are carried all through the organism and are also brought to the skin.

Tubercle bacilli gain entrance into the skin hematogenously in lupus, scrofuloderma, papulonecrotic and other forms of tuberculosis of the skin.

3. *Per continuitatem*, i.e., spreading by extension. In these cases the tuberculous process gradually extends from the affected organ to the surrounding tissues and finally involves the skin of the given area. Such spread of tuberculosis is particularly often observed in tuberculous affection of lymph nodes and bones. The causative agent of tuberculosis most commonly gains entrance into the skin in this manner in scrofuloderma.

4. *Autoinoculation.* If a patient has active, severe tuberculosis of the internal organs with a plentiful discharge of tubercle bacilli, the latter may gain entrance into the skin or the mucous membranes. For example, a patient with active tuberculosis of the lungs running an unfavourable course may develop tuberculosis of the oral mucosa, the skin of the lips and the nose. Tuberculosis of the kidneys may lead to tuberculosis of the skin and mucous membranes of the genitalia.

Tuberculosis of the skin occurs much less frequently than does tuberculosis of the lungs and of some of the other organs. The reason for it is that the skin offers comparatively less favourable conditions for the development and multiplication of the causative agent of tuberculosis.

Tuberculosis of the skin has many different clinical forms due to the differences in the general condition of the organism, indi-

vidual characteristics of its reactivity and various degrees of resistance to the tuberculous infection.

All clinical forms of tuberculosis may be divided into 2 groups, depending on the reactivity of the patient's organism.

The first group of tuberculous affections of the skin includes: (1) lupus vulgaris or true tuberculosis of the skin, (2) scrofuloderma, (3) tuberculosis verrucosa, and (4) miliary ulcerative tuberculosis of the skin and mucosa. The diseases of this group are characterised by slow development of the process, its chronic course, tendency to necrosis, histological structure simulating that of infectious granuloma and continuous presence of tubercle bacilli in the pathologic tissue of the focus.

The second group of tuberculous diseases of the skin includes: (1) papulonecrotic tuberculosis, (2) tuberculosis lichenoides and (3) indurative tuberculosis of the skin. These clinical forms of tuberculosis of the skin are marked by a benign chronic course and but little tendency to necrosis. In patients affected with these forms of tuberculosis tubercle bacilli are not always found or are found with difficulty in the foci of affection. Nor does the pathologic infiltrate always have the typical structure of infectious granuloma.

Lupus Vulgaris or True Tuberculosis of the Skin

Lupus is the most widespread form of tuberculosis of the skin. Patients with lupus vulgaris constitute 75-80 per cent of all cases of tuberculosis of the skin.

Lupus usually begins in childhood or youth, most commonly between 5 and 15 years of age, but may arise at any age. The main lesion of lupus vulgaris is the lupoma, the primary nodule of lupus. In the beginning the affection looks like a small—the size of a pinhead or lentile—purplish-red macule with a clear yellowish or brownish tint. Gradually the nodule begins to rise above the level of the surrounding skin. The nodule is soft. Upon pressure an eyed probe easily penetrates, as though "drops", into the infiltrate of the nodule, leaving an indentation which smoothes away but slowly ("probe" sign). Stronger pressure with the probe causes sharp pain and bleeding. If pressure is exerted on the lupoma with a glass spatula or a slide glass, the nodule becomes semitransparent yellowish-brown, resembling the colour of apple sauce ("apple sauce" sign). Gradually enlarging the separate nodules often coalesce forming continuous areas of lupous infiltrate. Lupomas develop slowly. Often lupomas show no appreciable changes for many months or even years.

In the process of their development lupomas always undergo necrosis and are replaced by scar tissue. In some cases the necrosis is "dry", the lupomas are resorbed without ulcerating and leave a cicatricial atrophy of the skin. In other cases the necrotic proc-

ess is more clearly marked, the lupomas become ulcerated and upon healing leave scars at their sites. The scars left by lupus have a characteristic appearance, i.e., they are superficial, thin, usually smooth and shiny, and easily wrinkle.

The areas of cicatricial atrophy are still thinner and more delicate; the skin in these areas easily forms very fine wrinkles and looks like crumpled cigarette paper.

Lupomas very often coalesce and form continuous patches, for which reason the scars forming after healing also cover rather large areas. Usually the borders of lupous scars are not clearly defined. Appearance of new nodules on the scars is a characteristic sign of lupus.

The arrangement of the nodules may be: (1) diffuse, in which case the nodules are localised in a circumscribed area and, coalescing, form a continuous infiltrate; (2) serpiginous, in which case the nodules gradually heal at one end of the focus, while new nodules continuously appear at the other end; (3) disseminated, i.e., characterised by formation of multiple nodules on various portions of the skin. The clinical picture of lupus vulgaris is very diverse owing to the number, sizes and arrangement of the nodules, and to the peculiarities of the course of the disease.

All the clinical varieties or forms of lupus may be divided into flat, or dry, and ulcerative. In dry forms of lupus the inflammatory phenomena are insignificant and ulcerations form rarely. The dry forms include *flat lupus* with flat barely elevated nodules varying in size from that of a pinhead to that of a hemp seed or lentile (Fig. 51). The nodules enlarge very slowly and may long persist without any particular changes. Subsequently flat lupus may develop into other clinical varieties of this disease.

There are also hypertrophic (tuberous), tumourlike and verrucose varieties of lupus (Fig. 52a).

Ulcerative forms of lupus are characterised by more clearly marked inflammatory phenomena and more intensive disintegration of the infiltrate. In a number of cases ulcerative lupus may develop from dry forms of the disease. In other cases the lupous infiltrate displays a tendency to ulcerative disintegration from the very outset. The ulcers are superficial, usually irregularly-shaped and with unclearly defined borders and uneven contours; they bleed readily and are not very painful (Figs. 52b, 53, 54a and 54 b). The floors of the ulcers are covered with flaccid, pale red granulations and a small amount of thin pus. These ulcers run a protracted course and heal with difficulty. Depending on their course, treatment and concurrent pyogenic infection, the ulcers may be covered with purulent or sanio-purulent crusts. Papillae sometimes grow on the floors of the ulcers (lupus vegetans).

If the lupous infiltrate spreads to deep tissues (muscles, cartilages, bones), deep ulcers are formed; sometimes these ulcers

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Fig. 51. Flat (exfoliative) lupus with destruction
of the tip of the nose and the alae nasi
(from P. Nikolsky)
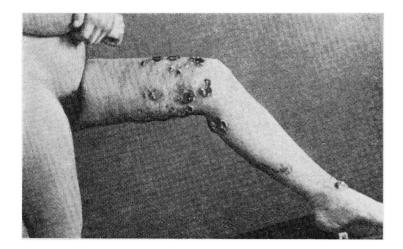
Fig. 52a. Verrucose and ulcerative lupus

Fig. 52*b*. Hypertrophic, partly ulcerated lupus
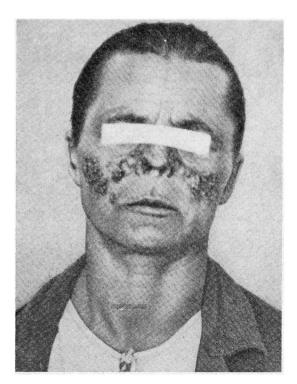
Fig. 53. Ulcerative lupus

Fig. 54a. Hypertrophic and ulcerative lupus

Fig. 54b. Tumourlike and ulcerative lupus

Fig. 55. Mutilating lupus of the hand

inflict considerable damage (disfiguring or mutilating lupus) (Fig. 55).

Lupus most commonly affects the face (in 75 per cent of all cases), the nose, especially its tip and alae, being particularly frequently affected.

The typical result of the lupous process — disintegration of the infiltrate and cicatrisation — is certain destruction of the tip and wings of the nose (apex and alae nasi). The nose of a lupus patient assumes the form of a "bird's bill" or a "sheep's nose".

If lupus is localised on the lips, cicatrisation may lead to deformation and constriction of the mouth. Localisation on the cheeks not infrequently results in eversion of the lower eyelids (ectropion).

About 25-33 per cent lupus patients have lesions in the mucous membranes. The mucosa of the nose, mainly of the cartilaginous part of the nasal septum and the alae nasi, is most commonly affected. The oral mucosa, predominantly the gums, are also frequently affected. Usually the process extends to the mucous membranes from the skin, but sometimes lupus begins on the mucous membranes and then spreads to the skin. On the mucous membranes lupus very often assumes an ulcerative form. Formation of crusts, sometimes massive and hindering respiration, is characteristic of lesions in the nasal mucosa.

Lupus is characterised by its prolonged, chronic course. In the absence of proper treatment it may persist for many years and even decades. The general condition of lupus patients may be satisfactory, although aggravation of pulmonary tuberculosis is often observed, especially in patients with chronic lupus. The protracted and stubborn character of the disease and its frequent localisation on the face with subsequent disfigurement greatly distress the patient.

Scrofuloderma (Tuberculosis Colliquativa)

Another very common from of tuberculosis of the skin, second to lupus in incidence, is scrofuloderma. Scrofuloderma constitutes 10-15 per cent of all cases of tuberculosis of the skin. This dis-

ease most frequently occurs in children and adolescents 5-15 years of age and is rarely observed in people past 30. The favourite localisation of scrofuloderma is the neck, less frequently the region of the sternum and inguinal folds, although it may develop on any part of the skin. The tubercle bacilli most commonly gain entrance into the skin in this form of tuberculosis by extension of the process from lymph nodes or bones affected with tuberculosis to the skin above them.

The disease begins with formation of a dense, painless node varying in size from that of a pea to that of a hazelnut in the subcutaneous tissue. The node gradually enlarges and adheres to the skin, the latter becoming purplish-red. Then the centre of the node softens and begins to throb, the node opens and through one or several small orifices discharges thin pus containing blood and fragments of necrotic tissue (Fig. 56). The orifices enlarge, coalesce, and finally give rise to an oval or irregularly-shaped ulcer with thin, soft, loosened, purplish-red edges overhanging the ulcer and forming deep pockets. The floor of the ulcer is uneven; it consists of granulations, readily bleeds and is covered with a purulent film. Scrofuloderma ulcers are scarcely painful, but if not properly attended become covered with heavy, purulent or sanio-sanguineous crusts. They may coalesce. Upon healing the ulcers leave scars of a characteristic appearance, i.e., they are superficial and often have projections and "papillae" of normal skin along their edges. The opposite ends of the scars are often joined by "bridges" of normal skin, under which it is possible to pass a probe.

Scrofuloderma runs a protracted course. In untreated cases several months may elapse from the appearance of a node in the subcutaneous cellular tissue to the cicatrisation of the ulcer. The total duration of the disease and the number of nodes and ulcers vary with the individual reactivity of the patient's organism (Figs. 57, 58 and 59). Scrofuloderma is a comparatively benign form of tuberculosis of the skin. In most cases it scarcely affects the patient's general condition. The ulcers tend to heal spontaneously even in the absence of treatment. The disease is often

Fig. 56. Scrofuloderma. Open node (from M. Bremer)
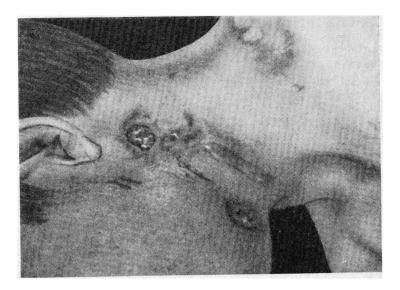
Fig. 58. Scrofuloderma
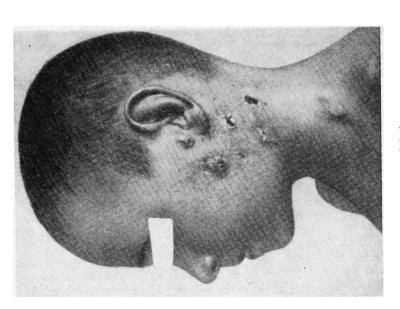
Fig. 57. Scrofuloderma

combined with other forms of tuberculosis of the skin and tuberculosis of other organs, especially tuberculosis of the lymph nodes. Scrofuloderma patients often have a typical "scrofulous appearance" — pale and puffy face, and swollen lymph nodes in the neck.

Tuberculosis Verrucosa

This rather rare form of tuberculosis of the skin develops either endogenously (most commonly hematogenically) or by infection from without. Infection from without most frequently occurs in workers of slaughterhouses and meat-packing plants, butchers and tanners who handle meat and hides of tuberculous cattle. In these cases the causative agent of the disease is the *Mycobacterium tuberculosis var. bovis.*

Tuberculosis verrucosa is also observed in workers of dissecting-rooms and morgues engaged in postmortem examinations of tuberculosis patients.

The disease begins with formation of a dense, painless, purplish-red or brownish-red tubercle the size of a hemp seed. The

Fig. 59. Multiple scrofuloderma

central part of the surface of the tubercle soon becomes covered with layers of horny material, while on the periphery the tubercle is surrounded by a narrow, red areola. Subsequently horny elevations, warty growths and crusts with deep fissures and cracks between them form on the surface of the tubercle. An infiltrated purplish-red fold is observed on the periphery of the warty formations (Fig. 60). The disease runs an exceptionally protracted, chronic course. The tubercles heal extremely slowly, nearly always without ulceration, and leave thin, soft scars. Tuberculosis verrucosa often simulates lupus verrucosus, but differs from it by the absence of the "apple sauce" sign and by a much denser infiltrate.

Fig. 60. Tuberculosis verrucosa (from A. Zenin and N. Torsuyev)

TREATMENT OF TUBERCULOSIS OF THE SKIN

Tuberculosis of the skin is a manifestation of general tuberculous infection. The treatment must strengthen the patient, increase the resistance of the organism, and create conditions unfavourable to the existence and multiplication of the causative agent of tuberculosis.

For the treatment of tuberculosis of the skin it is particularly important to establish for the patient an appropriate regimen, namely, adequate rest and sleep, daily long hours outdoors and rational (without overstrain) physical exercise. Patients affected with tuberculosis of the skin are prescribed a vitamin-rich diet with enough proteins and fats and somewhat limited amounts of carbohydrates and salt.

Medicine now has at its disposal effective agents for the treatment of tuberculosis of the skin. These agents include phthivazide and other isonicotinic acid preparations, streptomycin, dihydrostreptomycin, vitamin D_2 and PAS (paraaminosalicylic acid).

Phthivazide is one of the main antibacterial agents used in the treatment of tuberculous patients; it is administered per os in doses of 0.5-2 g (most commonly—1-1.5 g) per day. The daily dose is divided into 3-4 parts. A course of treatment requires administration of 80-200 g of the preparation. Phthivazide is usually well tolerated, but the patients must always be carefully watched during the treatment because of the possible side effects (cardiac pain, etc.).

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Streptomycin is administered intramuscularly in a dose of 0.25 g twice a day in the hospital and 0.5 g once a day to outpatients. A course of treatment requires 40-80 g of the preparation. The daily dose of dihydrostreptomycin is 0.5-1 g. Streptomycin treatment often gives rise to complications—toxicoderma, impaired hearing, tinnitus aurium and headache. In such cases the treatment with streptomycin is discontinued.

Vitamin D_2 is administered to adult lupus and scrofuloderma patients per os in a dose of 50,000-100,000 u per day, depending on the character of the disease, the age and general condition of the patient. The treatment is continued over a period of 5-8 months, the patient's general condition and tolerance of the treatment being carefully watched because of the danger of serious complications during the treatment. Vitamin D_2 produces a good therapeutic effect on lupus patients, especially in cases of ulcerative lupus and lupus of the mucous membranes.

PAS (paraaminosalicylic acid) is prescribed mainly for patients with scrofuloderma and certain other forms of tuberculosis of the skin. It is administered per os in a dose of 8-12 g per day, 500-1,000 g per course of treatment.

Antituberculous preparations produce the best effects when administered complexly, i.e., when two or even three of them are administered simultaneously. For example, good effects are produced by simultaneous daily administration of 1-1.5 g of phthivazide and 0.5 g of streptomycin or 0.5-1 g of dihydrostreptomycin.

For most patients with tuberculosis of the skin one course of treatment is not enough because of frequent relapses of the disease. It is therefore desirable to administer repeated courses of treatment, in which case a change in the preparations is indicated. General roborants—ascorbic acid, vitamin B_{12}, fish liver oil, and preparations of iron, arsenic and phosphorus—are used in the general treatment of tuberculosis of the skin, depending on the patient's general condition.

A favourable effect on patients with tuberculosis of the skin, especially with lupus and scrofuloderma, is produced by sunbaths.

To prevent aggravations of tuberculosis of the lungs, lymph nodes and other organs, the patient's general condition must be carefully watched. Active tuberculosis of the lungs or other organs contraindicates treatment with sunbaths. Treatment at health resorts and kumys therapy are very effective in tuberculosis of the skin.

The *physiotherapeutic methods* used in the treatment of tuberculosis of the skin are mainly carbon arc and mercury vapour lamps.

Topical, external treatment is administered mainly to patient with lupus and scrofuloderma. It is necessary primarily in cases where the foci of affection on the skin and mucous membranes do not heal rapidly enough under general treatment.

Topical treatment is aimed at destroying tuberculous tissue, changing it to a wound surface and expediting its cicatrisation, for which purposes chemical agents, physiotherapy and surgery are used.

The chemical agents of destructive therapy include ointments and pastes which selectively destroy tuberculous tissue. These are arsenic "Plantagin paste", ointment with 10-50 per cent pyrogallol, and certain other agents.

The physiotherapeutic methods used for destroying tuberculous tissue are diathermocoagulation, galvanocautery and sometimes carbon dioxide snow.

The surgical methods employed in destroying tuberculous tissue are excision of a circumscribed, isolated focus of lupus, and curettage of scrofuloderma nodes and sometimes of lupous foci with a sharp curet. After the curettage the wound surface is covered with a layer of streptocide water paste or is cauterised with a silver nitrate stick.

Topical treatment can be effective only when combined with general treatment of tuberculosis of the skin.

CONTROL OF TUBERCULOSIS OF THE SKIN IN THE U.S.S.R.

The Soviet Government and Soviet public health services are sparing no efforts in controlling tuberculosis of the skin. All new cases of tuberculosis of the skin are registered without fail, there is a network of skin tuberculosis offices in anti-tuberculosis dispensaries, and the patients are actively treated and followed up by these dispensaries. These measures have sharply reduced the incidence of tuberculosis of the skin in the U.S.S.R. The experience of the Soviet public health services attests that the incidence of tuberculosis of the skin can be reduced by providing all patients with free, qualified medical aid and active dispensary service.

LUPUS ERYTHEMATOSUS

The etiology of lupus erythematosus is still unknown. The formerly widespread conception of tuberculous etiology of this disease is now rejected by most investigators. Nor has the etiologic role of streptococcus or virus infection been demonstrated.

An important part in the pathogenesis of lupus erythematosus is played by connective tissue dysfunction, endocrine disorders and foci of chronic infection (carious teeth, diseases of the gums, tonsils, paranasal sinuses, etc.). Most lupus erythematosus patients exhibit increased sensitivity to sources of radiant energy, especially sunlight. This explains why lupus erythematosus most commonly begins and becomes aggravated in spring or summer.

Women are affected with lupus erythematosus more frequently than men. The disease usually begins between 18 and 50 years of age.

The chronic form of lupus erythematosus is the most common. In this form of the disease pink-red and saturated-red round or oval macules with clearly defined borders appear on the exposed parts of the body, most frequently on the face and more rarely on the pinnas of the ears, the scalp, neck and hands. The skin in the area of the macules becomes gradually infiltrated, the lesions grow larger, coalesce and form patches. Continuous infiltrated foci are often formed; on the face these foci may resemble butterflies.

In most cases there is a large number of closely adhering whitish or greyish lamellar scales on the surface of the patches. Scraping of the patches is somewhat painful. When the scales are removed horny spines can be seen on their undersides (Figs. 61 and 62).

The chronic inflammatory process in lupus erythematosus terminates in atrophy of the skin, for which reason there are usually areas of thin atrophic skin in the centres of the patches, a zone of redness, infiltration and scaling closer to the periphery, and, lastly, a bright-red areola along the edges of the patches. The vermilion border, usually of the lower lip, is also frequently affected (Fig. 63). Intense redness, marked hyperkeratosis, scaling of the foci and resultant atrophy considerably disfigure the face and thereby inflict psychic trauma on the patients.

Lupus erythematosus usually runs a chronic course, remissions are followed by aggravations and appearance of new foci, most commonly in spring and summer.

Systemic lupus erythematosus is observed less frequently. This form of the disease may be subacute, acute and chronic. The
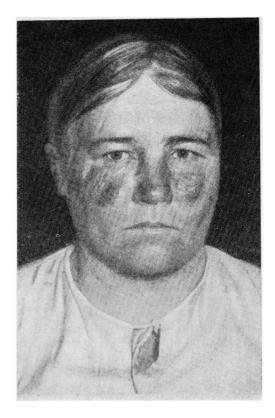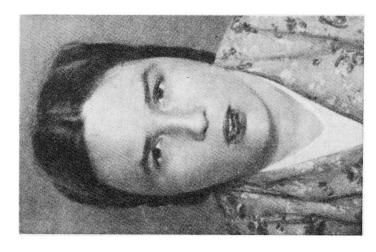
Fig. 61. Lupus erythematosus

subacute form is characterised by appearance of disseminated foci, including foci on covered parts of the body, pains in the joints, moderately elevated temperature and anemia. An acute form of this disease occurs still less frequently. Acute lupus erythematosus is an extremely severe disease with various eruptions on the skin and mucous membranes, affection of the kidnyes, liver and heart, high and often septic temperature, pains in the joints, anemia and, in most cases, lethal results. Chronic systemic lupus erythematosus is marked by affections of the internal organs, joints and nervous system. The skin is often entirely unaffected or exhibits macular eruptions.

Treatment. Lupus erythematosus patients must be thoroughly examined, especially for chronic infectious foci, tuberculosis of the lungs and other organs, and must be given corresponding treatment. The best treatment is with resochine in a dose of 0.25 g once or twice a day for several weeks.

Fig. 63. Affection of the vermilion border in lupus erythematosus
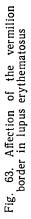
Fig. 62. Lupus erythematosus (affection of the face and hairy part of the head).

Acrichine (quinacrine) is also prescribed, usually in a dose of 0.1 g 3 times a day for 10-day periods at 5-10-day intervals (a total of 3-5 periods). Nicotinic acid is administered simultaneously with acrichine.

Good effects are produced on many patients by vitamin B_{12} administered in doses of 100-500 micrograms daily or on alternate days. Some patients benefit from injections of 0.1-0.2 ml of crisanol (oil suspension of calcium aurothiopropanosulfonate and calcium gluconate) once in 3-7 days, a total of 20-25 injections.

The external treatment of lupus erythematosus consists in applications of anti-inflammatory and keratolytic agents—ichthyol, sulfur, boric acid and salicylic acid in the form of ointments and pastes. The more recent the eruptions and the brighter the redness, the weaker must the concentration of these substances be.

A 10 per cent acrichine plaster or a 10 per cent ointment containing acrichine may be effectively applied to the patches with considerable infiltration and in the absence of phenomena of aggravation. The acrichine ointment (or plaster) is applied to the foci of the disease and is covered for 2-4 days with an "imbricated" adhesive plaster. If the removal of this plaster reveals phenomena of irritation, indifferent lotions, pastes and ointments are applied to abate them, and after the abatement an acrichine plaster or ointment is reapplied. This procedure is repeated several times until the infiltrate is resorbed. It is extremely important to protect the patient's skin and, especially, the foci of affection from sunlight and other sources of radiant energy. The following protective ointments are used for this purpose: ointment containing 15 per cent paraaminosalicylic acid, ointment with 4 per cent paraaminobenzoic acid, and ointments containing 10 per cent quinine, tannin and salol (phenyl salicylate) each. The patients must wear broad-brimmed hats or carry umbrellas to protect the face from the sun.

The main agents for the treatment of systemic acute, subacute and chronic forms of the disease are blood and plasma transfusions, corticosteroid hormones (see p. 68), a high protein diet and vitamin B_{12}.

LEPROSY

Leprosy is a severe, chronic infectious disease; it affects the entire human organism and is accompanied by changes in the skin and mucous membranes.

Leprosy has been known since hoary antiquity. The spread of leprosy was favoured by the crusades, wars and distressing economic conditions of the population. Before the Great October Socialist Revolution there were several foci of leprosy on the territory of Russia—in the lower reaches of the Volga and Don rivers, along the Baltic coast and in Central Asia. It was very difficult to control leprosy because of the inadequate knowledge of the etiology, pathogenesis and epidemiology of the disease and the too little attention paid by the reactionary tsarist government to public health. Today leprosy is widespread in India, Africa, South America, Indonesia and certain other countries.

The causative agent of leprosy is the *Mycobacterium leprae.* In the smears of mucus taken from the nose, in the discharge from the ulcers and in the tissues of the affected organs the causative agent has the form of bacilli often arranged in clusters resembling globi or cigar-bunches.

The disease is transmitted through close everyday contact of a healthy person with a leper and in unsatisfactory sanitary conditions. Children are much more susceptible to leprosy than are adults. Infection with leprosy apparently most commonly occurs in childhood.

The bacteria causing leprosy gain entrance into the human organism through the nasal mucosa or through the skin.

The incubation period of leprosy is from 1 to 5-10 years and even longer. In most patients the first signs of the disease appear 3-5 years after the infection has taken place. Prodromal symptoms— dryness in the nose, nasal hemorrhages, swelling of inguinal and femoral lymph nodes, weakness, headaches and fever—may be observed at the end of incubation. The prodromal period lasts from a few days to several months.

The clinical manifestations of leprosy vary very widely because changes produced by the disease may develop in any organ. The skin and mucous membranes are affected particularly frequently. Three forms of leprosy are distinguished according to the clinical picture of the disease—lepromatous, tuberculoid and indeterminate.

The *lepromatous type of leprosy* is characterised by a severe course. The patients affected with this type of leprosy show very

low resistance to its causative agent; they develop lesions in the skin, mucous membranes, the eyes, lymph nodes and internal organs. Lepromas—red-brownish tubercles or nodes with a smooth surface and "fatty lustre"—appear on the skin. At first the tubercles are the size of a lentile or a pea, and the nodes are the size of a hazelnut or a plum. Gradually enlarging the lepromas may coalesce into vast continuous infiltrates. Erythematous, hyperpigmented and depigmented macules appear in addition to the tubercles and nodes (Fig. 64). The tubercles, nodes and diffuse infiltrates may gradually be resorbed, but they scale and the skin subsequently atrophies. The infiltrate frequently softens and slowly healing ulcers form, leaving scars after healing. Disintegration of the lepromas leads to mutilation of the face, destruction of the fingers and other deformities.

Lepromas form on any part of the skin. Especially characteristic are lesions on the face—continuous infiltrates and separate tubercles form on the forehead, in the region of the eyebrows, on the ear flaps and lips. The skin of the face becomes uneven, deep folds are formed and the eyebrows fall out. The infiltrated skin of the forehead and the infiltrates on the cheeks, nose and lips impart a leonine appearance to the patient's face (Fig. 65). Reduced sensitivity is observed in the region of the lepromas and macules. The mucous membranes of the nose, mouth and larynx are often affected. As a result of changes in the larynx, the patient's voice becomes hoarse and nasal; sometimes the patient loses his voice altogether. The disease affects lymph nodes, bones and eyes. The most frequently involved internal organs are the spleen, liver, testes and their adnexa.

The lepromatous type of the disease not only runs the severest course, but is also the most contagious form of leprosy. The nasal mucus of 95 per cent of the cases of this form of leprosy contains large numbers of the *Mycobacterium leprae*.

The *tuberculoid type of leprosy* is the most favourable and benign as to its manifestations and general course. Patients affected with this form of the disease have rather high resistance to leprous infection. The *Mycobacterium leprae* is rarely found in their nasal mucus. The disease is not very contagious. The eruptions on the skin appear in the form of nodules, macules and, more rarely, tubercles (Fig. 66). The nodules are often arranged in the form of rings, arches and garlands in the centre of which cessation of perspiration and diminished cutaneous sensitivity, to the point of its complete loss, are observed. The eruptions heal by slow resorption, leaving a depigmentation or atrophy. The affections of the nerves occur as thickenings of the nerve trunks, pareses and paralyses of individual nerves.

The *indeterminate type of leprosy* is characterised by unstable, variable resistance of the organism to the causative agent of
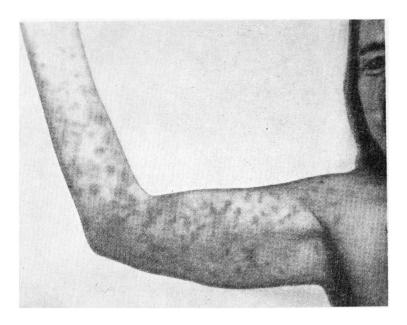
Fig. 64. Maculous eruption in leprosy
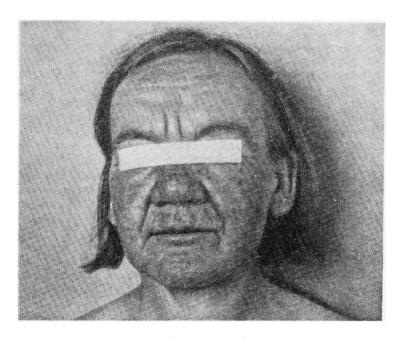
Fig. 65. Lepromatous leprosy
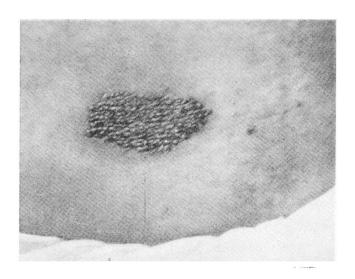
Fig. 66. Tuberculoid leprosy (from N. Torsuyev)

leprosy. Considerable variations in the resistance of the organism may change the picture of the disease, and the indeterminate form may develop into the lepromatous or tuberculoid forms.

The indeterminate type of leprosy is marked by affection of the nerves and skin. The eruptions on the skin are erythematous or depigmented macules of various sizes and shapes. Sometimes hyperpigmented macules are also observed. The macules always exhibit considerable diminution in sensitivity which is sometimes completely lost.

In indeterminate leprosy the affections of nerves are particularly pronounced. A thickening and calcification of nerve trunks, diminished sensitivity and motor disorders due to paralysis of the nerves and atrophy of the muscles, especially those of the face, hands and feet, are very often observed. The affections of the nerves often involve other trophic disorders, namely, formation of chronic ulcers, deformation of joints and destruction of bones.

The course of leprosy is chronic and depends on the general condition of the organism. In some cases the disease rapidly progresses, is accompanied by extensive destructions and results in the patient's death from cachexia or concurrent pulmonary tuberculosis and other diseases. In other cases leprosy may persist for decades with the patient in a satisfactory general condition (in the tuberculoid and sometimes in the indeterminate forms of the disease).

Treatment. Early forms of leprosy are easier to treat. Far advanced leprosy yields to treatment with greater difficulty. Early

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diagnosis and early treatment are therefore enormously important. For successful treatment patients require an appropriate regimen—sufficient sleep and rest, long hours outdoors, adequate nutrition rich in animal proteins, fats, carbohydrates and vitamins; the diet must include milk, eggs, meat and fruit.

Moderate work under a physician's observation is beneficial for lepers. The main drugs are sulfonamides (sulfetron, etc.). These preparations produce good results in many cases of leprosy.

The most popular old medicinal substance is chaulmoogra oil which is administered subcutaneously, intracutaneously and intramuscularly, and, less frequently, in drops per os.

Control of leprosy in the U.S.S.R. In Soviet time the leprosy incidence in the U.S.S.R. has considerably diminished as a result of planned control of this disease by the Soviet public health services.

One of the most important measures of leprosy control is isolation of patients with active disease symptoms in special therapeutic and work colonies (leprosoriums). The other lepers may take treatment as outpatients provided they are under regular medical observation.

Children born of leprous mothers are separated from the latter and are nursed artificially. All members of a family affected with leprosy are periodically given medical examinations.

Lepers must keep their bodies, clothing and dwellings thoroughly clean. Before washing the underwear of lepers must be disinfected by soaking for 1 hour in a 5 per cent lysol solution or carbolic acid.

SKIN DISEASES CAUSED BY ANIMAL PARASITES

Scabies

Scabies is a contagious skin disease caused by the scab or itch mite (*Sarcoptes scabiei*). The spread of scabies is favoured by poverty and inadequate health education of the population, for which reasons in some countries, especially the colonial and dependent countries, scabies often affects masses of people. In tsarist Russia scabies was one of the most common skin diseases. The enormous changes in the life of the people which have occurred since the Great October Socialist Revolution, the rise in living standards and the flourishing of socialist culture have brought about a sharp decrease in the incidence of scabies.

Scabies is a very rare occurrence in the U.S.S.R. today. Every medical worker—physician's assistant, midwife and medical nurse —must be able to identify this disease, treat scabies patients and carry out preventive measures.

The causative agent of scabies—the itch or scab mite—parasitises chiefly on man. The mites parasitising on the skin of various animals rarely affect man and, if they do, the affections pass off in most cases rather quickly even without treatment.

The itch or scab mite belongs to the class of Arachnida. It is shaped like a turtle (Fig. 67). The female mite is 0.35 mm long,
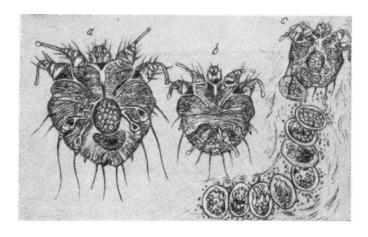
Fig. 67. Scab mite (from P. Nikolsky)
a—female; b—male; c—burrow of the scab mite with the mite, eggs and excrements

the male—about 0.25 mm. Upon coming in contact with the human skin the female mite burrows into the stratum corneum in the way the mole does underground. It takes 7-12 days for the symptoms of scabies to develop after the mite has contacted the human skin. The male mite does not burrow into the skin, but lives in its recesses under the scales. Young mites emerge within 3-4 days from the eggs laid by the female and within several weeks attain sexual maturity.

Itching is a constant symptom of scabies. It increases in the evening and at night when the mites become more active and burrow into the skin. In the daytime the mites are for the most part inactive. The itching varies in intensity and depends on the state of the patient's nervous system. In some cases the itching is so intense that the patients cannot even sleep; other patients scarcely complain of itching.

In scabies the skin eruptions are localised on the flexor surfaces of the upper extremities, on the chest, abdomen, buttocks, male genitals and thighs, and on women's breasts. The eruptions are particularly frequently observed in the interdigital folds of the hands and on the flexor surface of the wrists.

Scabies does not affect the skin of the head and very rarely affects the neck, back and shanks.

The most typical lesion of scabious eruptions is the *cuniculus* which looks like an arched or winding greyish or blackish line 2-3 mm long. Examination of a cuniculus through a magnifying glass shows it to consist of closely arranged black dots—the "shafts" which the female mite digs for the access of air and the emergence of young mites to the surface of the skin. Cuniculi can most commonly be seen in the interdigital folds and on the flexor surface of the wrists. They are hard to find on other parts of the body. In nurslings the cuniculi are well marked on the palms and the soles of the feet.

In addition to cuniculi the patient's skin exhibits reddish nodules, vesicles and sanguineous crusts the size of a pinhead. The intense itching makes the patients scratch, which leads to complications, namely, staphylococcal impetigo, folliculitides and furuncles. The scratches or improper treatment may complicate scabies with dermatitis (Fig. 68).

Secondary pyoderma and dermatitis often cause enlargement of lymph nodes.

The diagnosis of scabies is established on the basis of the itching and presence of cuniculi in the interdigital folds of the hands and on the flexor surface of the wrists, reddish nodules, sanguineous crusts, and sometimes small vesicles on the surface of nodules or on unaffected skin. Very tidy persons who keep their hands particularly clean and workers who have to do with petroleum products, tar and turpentine may not have any typical
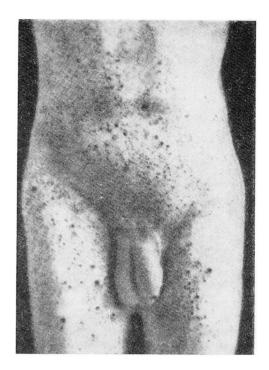
Fig. 68. Scabies (from M. Zheltakov)

cuniculi. In the absence of typical cuniculi the diagnosis of scabies is established on the basis of the itching and the presence of an eruption consisting of small nodules, sanguineous crusts and secondary pyoderma, if all these lesions have a characteristic localisation, i.e., are localised on the chest, abdomen, anterior part of the axillae, on the buttocks, medial surface of the thighs and on the male genitals.

If the patient is not given proper treatment the disease may continue for an indefinite period of time (many months).

Scabies may be transmitted through close contact of a healthy person with a patient (by sharing a common bed), especially at night—the time of the greatest activity of the itch mite. Another way of contracting the disease is by using things used by the patient—bedding, linens, clothing, towel and sponge. The disease may also be contracted in baths if the latter are kept in an unsanitary condition. Children may contract the disease from each other while playing, wrestling, etc.

Treatment. All methods of treating scabies are based on rubbing into the skin of substances which loosen and exfoliate the stratum corneum and kill the mite. Soviet scientists have made

a cosiderable contribution to the treatment of scabies by elaborating new methods of treatment.

Demianovich's method of treating scabies is the fastest and most effective. According to this method, the patient must wash at home with hot water and soap directly before the beginning of the treatment. Then the patient completely undresses and over a period of 10 minutes rubs a 60 per cent sodium hyposulfite solution (Solution No. 1) into every part of his skin. After a 10-minute interval the same solution is rubbed in again for another 10 minutes. The solution must be poured into a plate, bowl or basin. The patient immerses his hands in the solution and vigorously rubs it into the skin. As this is done hyposulfite crystals are formed. The patient must not shake them off the skin, but must continue to rub the solution in together with the crystals thereby loosening the stratum corneum and achieving deeper penetration of the solution.

After the second embrocation of the hyposulfite solution another 10-minute interval is made to let this solution dry. Following this the patient begins to rub the second solution (Solution No. 2)—a 6 per cent concentrated hydrochloric acid solution (or a 20 per cent commercial diluted hydrochloric acid solution)—into the skin. The solution is poured in small portions from the bottle on the palm and is vigorously rubbed into the skin over a period of 5 minutes, which is followed by a 5-minute interval. This is repeated 4 times with 5-minute intervals also to let the solution dry.

Demianovich's method is based on the interaction of sodium hyposulfite and hydrochloric acid with the resultant formation of sulfur dioxide and free sulfur. Both these substances possess scabicidal properties.

The reaction between sodium hyposulfite and hydrochloric acid takes place both on the surface of the skin and in the stratum corneum into which sodium hyposulfite and hydrochloric acid are rubbed in.

After the last embrocation of hydrochloric acid the patient lets his skin dry for 5-10 minutes and puts on clean underwear. The entire treatment by Demianovich's method takes 1 hour and 20 minutes or a little longer.

The patient must wash and change his underwear no sooner than 3 days after the treatment and must report to a medical worker for a check-up. If the patient exhibits any scabious phenomena, the treatment by Demianovich's method is repeated.

Failure to observe the rules of the treatment suggested by M. Demianovich defeats the purpose and leads to relapses of the disease, for which reason the patient must be given detailed instructions. If the patient confuses the solutions and first rubs in the hydrochloric acid and then the sodium hyposulfite he will not effect a cure.

Another method of treatment is application of Wilkinson's ointment which consists of 15 per cent sulfur and pitch each, 10 per cent chalk, 30 per cent green soap and 30 per cent fatty base. Before beginning the ointment treatment the patient must, as in the treatment by Demianovich's method, wash and change his underwear. The ointment is rubbed into every part of the skin (except the head and neck) every evening (for 4-6 evenings). It is rubbed particularly carefully into the interdigital folds, the flexor surface of the wrists, the chest, abdomen, buttocks and thighs. The shortcoming of this method is that Wilkinson's ointment may produce an irritation of the skin.

A 10-30 per cent sulfur ointment is often used in the treatment of scabies. Children and persons with a thin, delicate or dry skin are prescribed weaker concentrations of sulfur ointment. Sulfur ointment affects the mite somewhat less than does Wilkinson's ointment, but it also produces complications less frequently. On the seventh day the patient washes and changes his underwear.

K. Diakov's sulfur soap may be used in the treatment of scabies. It consists of 125 g of sulfur, 50 g of soap, 25-50 g of starch or flour and 350 ml of water. This soap is daily (for 4-5 days) rubbed for 15-20 minutes into the skin of the entire body. After the soap has dried the patient puts on his underwear; the following day he washes off the dry soap and rubs in the soap again. If the phenomena of secondary pyoderma or dermatitis are very strongly pronounced, the treatment should be postponed 1 or 2 days to eliminate the extant complications.

The preliminary treatment of complicated scabies must not be delayed more than 1-2 days because the itching and scratching caused by scabies support the phenomena of pyoderma and dermatitis. In such cases it is necessary to begin the treatment of scabies despite the presence of certain phenomena of pyoderma or dermatitis. For patients with complicated scabies it is best to prescribe treatment by Demianovich's method or inunction of ointments with weaker concentrations of antiparasitic agents, namely, a 10 per cent sulfur ointment or half-and-half Wilkinson's ointment and zinc ointment. Weaker ointments require longer application, but make it possible to avoid excessive irritation of the skin.

Ointments with weaker concentrations of antiparasitic agents are also prescribed for the treatment of scabies in children; these are, for example, half-and-half Wilkinson's ointment and zinc ointment or 1 part Wilkinson's ointment and 2 parts zinc ointment, or 5-15 per cent sulfur ointment.

In patients with a thin, delicate or dry skin antiscabious treatment may cause dermatitis medicamentosa. The patient's skin becomes red and dry and begins to scale and itch; in some cases

dermatitis is of a more acute character and is accompanied by edema, vesicles and exudation.

Prevention of scabies must be aimed primarily at ensuring careful and accurate treatment of patients and at preventing relapses of the disease. For this purpose dispensaries keep records of the patients. If the patient fails to report for a check-up after the treatment, he is summoned to the medical institution or a community nurse visits him at home for the purpose of checking up on the results of the treatment.

Disinsection of the patient's underwear, clothing and bedding is very important for preventing relapses of scabies. The underwear and linens changed by the patient before the beginning and after the end of the treatment is either disinsected in a dry or moist hot chamber, is dusted with DDT powder or is boiled. The clothing is either disinfested in a disinfection chamber, is dusted with DDT or pressed with a hot iron.

Scabies is a contagious disease. It is therefore necessary to examine all members of the patient's family and the other persons who were in close contact with him. If the disease is contracted by a school, kindergarten or nursery child, or by a worker of a children's institution, it is necessary to examine all the children and personnel of this institution. Immediate isolation and treatment of all patients discovered during the examination makes it possible quickly to liquidate the disease and prevent it from spreading among the children.

In areas where cases of scabies are still observed medical workers must conduct a systematic antiscabies campaign among schoolchildren, teachers, personnel of other children's institutions, at parents' meetings, in clubs and hostels.

Pediculosis

Pediculosis or infestation by lice is a concomitant of poverty and low sanitary standards of the population. In some countries it is a widespread occurrence. The rise in the living standards and culture of the working people in the Soviet Union has made pediculosis a rare phenomenon in this country. Medical workers must regard complete eradication of pediculosis, wherever it still occurs their immediate task. They must therefore be able to recognise all manifestations and effects of pediculosis, and to take measures for exterminating lice.

Three species of lice parasitise on the human skin—the head louse, the body louse and the crab louse (Fig. 69). They are all parasitic only on man and are not found on any animals.

The *head louse* (*Pediculus humanus var. capitis*) — a dark-grey insect, 2-4 mm long, lives on the hairy part of the head, mainly in the region of the occiput and sinciput. The female louse
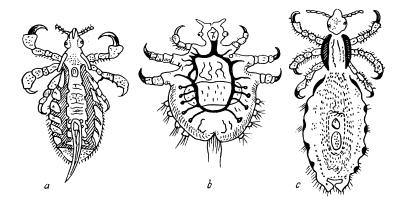
Fig. 69. Lice
a—head louse; *b*—crab louse; *c*—body louse (from P. Nikolsky)

is capable of laying during its lifetime up to 120-150 eggs (nits) which it fastens to the hair by means of a special chitinous substance. Young lice emerge from the nits within 5-6 days and within about 3 weeks are already capable of reproduction.

Head lice cause an intense itching of the skin in the hairy part of the head. The itching and resultant scratching lead to development of secondary pyoderma and dermatitis.

The skin in the hairy part of the head becomes red and covered with follicular pustules and erosions with a copious serous exudate and numerous purulent and serous crusts. Lice can often be found under the crusts. The cervical, occipital and parotid lymph nodes are often enlarged.

Head lice are transmitted through direct contact, headgear, kerchiefs, combs and hairbrushes.

Treatment. To treat pediculosis in children or men, it is best to shave or closely crop the hair on the head and then gently rub into the scalp a mixture of half kerosene and half vegetable oil. Pediculosis in women usually has to be treated without removing the hair. To do this, the hair is heavily impregnated with the same mixture of kerosene and vegetable oil, tied with a kerchief and left overnight. In the morning the hair is washed with hot water and soap. During the washing the hair is soaked several times in heated table vinegar (to dissolve the chitin) and is thoroughly combed with a fine comb.

Concurrent pyoderma or dermatitis should be treated as usual.

The *body louse* (*Pediculus humanus var. corporis*) is greyish and somewhat larger than the head louse. It feeds on human blood and lives in the folds and stitches of the underwear and lays its nits in the stitches and folds of the cloth, on long and downy hair

and in the folds of the skin. In cases of considerable infestation by lice nits can also be found in the outer garments and even on belts and buttons. Body lice can also live in bedding. The bites of body lice cause intense itching and sometimes eruptions of blisters and in some persons intensely itching nodules lasting several days. The itching caused by body lice makes the patients scratch their skin. In the favourite places of habitation of body lice—on the posterior surface of the neck, the loins, the scapulae and between them—the skin is usually covered with scratches. The scratches are often complicated by secondary pyoderma— staphylococcal impetigo, folliculitis, furunculosis and enlarged lymph nodes. In persons heavily infested with lice the skin becomes yellowish or brownish, especially in the area of the scapulae and loins, and exhibits numerous scratches, purulent lesions and resultant scars ("vagabond's disease"). Body lice are transmitted through contact with a person infested with them.

Treatment. The treatment of pediculosis corporis is based on establishment of a hygienic regimen and disinsection of the patient's underwear and clothing. Thorough washing with hot water and soap, and change of underwear usually rid the patient of body lice. In cases of neglected pediculosis the hair in the axillae, on the pubes, etc., is shaved off and these parts are painted with a 5-10 per cent water emulsion of freshly prepared soap K (soap containing insecticide bis-ethyl xanthogen). A good effect is produced by dusting with DDT the underwear and clothing freshly put on. The underwear and clothing may be disinsected by various methods—dusting with DDT, in a dry-heat or formalin vapour chamber, boiling with lye, or soaking in an emulsion or soap K.

The *crab louse* (*Phthirus pubis*) is flat, whitish and up to 1.5 mm in size. It lives mainly in the pubic region and in the areas of the genitals, the anus and on the abdomen. When crab lice are very numerous they parasitise on the thighs, chest, in the axillae and on any part of hair-covered skin, including even the eyebrows and eyelashes. The crab louse settles on the hair at the point of its emergence from the hair follicle, firmly attaching itself to the hair. It lays its nits also on the hair. Greyish or pale-blue macules the size of a lentil sometimes appear on the skin at the sites of habitation of crab lice. In most cases crab lice cause intense itching. The crab louse is most commonly transmitted during sexual intercourse.

Treatment. The treatment of pediculosis pubis consists in shaving off the hair at the sites of affection and subsequent washing with soap. Then, over a period of 2-3 days a mild mercurial ointment or a 10 per cent ammoniated mercury ointment is rubbed into the skin at the affected areas, or the skin is rubbed down with vinegar of mercury bichloride (1 : 300). Dusting with DDT or painting

the affected areas with a 2 per cent DDT emulsion in vegetable oil also quickly kills the crab lice.

Prophylaxis. Pediculosis is a sign of uncleanliness and ignorance of hygiene and is always a considerable danger to society because lice are vectors of a number of severe communicable diseases. Medical workers must do all they can to prevent pediculosis.

Upon discovery of pediculosis medical workers must: (1) take measures to rid the patient of it, (2) examine the members of the patient's family and the persons who were in contact with the patient, and (3) lecture on health education in the group or community where the case of pediculosis was discovered.

Individual prevention of pediculosis is based on consistent observance of the rules of hygiene, i.e., bathing or showering and changing the underwear at least once a week, boiling the underwear before it is laundered, and keeping the dwelling clean.

DERMÂTITIDES

Dermatitis or *contact dermatitis* is an inflammation of the skin caused by the action of an external irritant. There are external irritants which always cause an inflammatory reaction in all people when coming in contact with the skin. Such irritants are called obligate (unconditioned) or absolute irritants. They include strong acids, strong alkalis, hot water (70°C and higher), large doses of sunlight, ultraviolet and roentgen rays.

Other external irritants do not always induce irritation and not in all people at that. Their irritating effects manifest themselves only in patients who are hypersensitive to them. The irritants of this type are called facultative (conditioned) or relative. The increased sensitivity to conditioned irritants may be either inborn (idiosyncrasy) or acquired as a result of the patient's repeated contact with the irritant (allergy). That is why conditioned irritants play the part of *allergens*.

Artificial, or *simple*, and *allergic* dermatitides are distinguished according to the type of irritant and character of the response inflammatory reaction. There are also *toxicodermas*—inflammatory reactions of the skin in response to penetration of allergens into the organism through the digestive or respiratory tracts, or through subcutaneous, intramuscular or intravenous administration.

Of course, the irritating effects depend not only on the character of the irritant, but also on the state of the organism's reactivity. For example, two persons simultaneously exposed to equally long solar irradiation may develop solar dermatitis greatly differing in the intensity of the inflammation.

A. Polotebnov was one of the first to emphasise that the "skin of different people reacts differently to the same irritation".

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Artificial or *simple dermatitides* may be caused by various external influences—mechanical, thermal, actinic (from sources of radiant energy) and chemical.

Artificial dermatitides develop from the action of unconditioned irritants on the skin.

The dermatitides produced by mechanical causes, most commonly friction or pressure, include water blisters, calluses, dermatitides in skin folds, and chafing.

The dermatitides due to thermal influences include burns and frostbite. Prolonged influences of heat—application of hot water bottles and staying near a furnace or bonfire—lead to formation of stable pigmentation which looks like a net. Prolonged action of cold results in development of *chilblains*, i.e., purplish or brownish swellings form on the dorsal surfaces of the fingers and toes, on the ears and sometimes on other parts of the skin; on warming they cause a sensation of itching, burning and pain.

The dermatitides produced by the effects of radiant energy include solar dermatitides, dermatitides caused by ultraviolet and roentgen rays, rays of radium and radioactive isotopes.

The chemical substances causing dermatitides are very numerous; they include acids, alkalis, certain salts (blue vitriol, barium sulfide, etc.), and chemical weed- and pest-killers.

The dermatitides produced by chemical irritants also include dermatitides medicamentosa caused by external application of certain medicinal substances, for example, that of Wilkinson's ointment used in the treatment of scabies, or concentrated lactic and salicylic acid ointment (Fig. 70). The stronger the irritant and the longer its action, the more intense the inflammatory phenomena.

Dermatitis is characterised by redness, edema and swelling of the skin in the affected area. The skin is hot to the touch, and the patient complains of pain, burning and sometimes itching. In cases of intense inflammation vesicles and blisters are formed; the vesicles and blisters may open and produce an erosive surface (Fig. 70). The clinical picture of dermatitis may greatly resemble that of acute eczema.

To distinguish simple dermatitis from allergic dermatitis or eczema, the following must be taken into consideration:

1. Simple dermatitis develops immediately after the action of the irritant on the skin.

2. The localisation of simple dermatitis strictly corresponds to the area acted upon by the irritant.

3. The clinical picture of simple dermatitis is most commonly characterised by lesser polymorphism and greater uniformity of the lesions than it is in eczema.

4. In simple dermatitis the inflammatory phenomena abate soon after the action of the irritant has ceased.

Fig. 70. Medicamentous bullous dermatitis (caused by ointment containing salicylic and lactic acids)
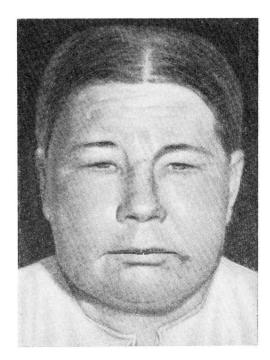
Fig. 71. Allergic dermatitis

On drying the exudate forms crusts. As soon as the action of the irritant has ceased, healing begins, the redness and edema diminish, no new vesicles and blisters appear, and the crusts gradually fall off. Lamellar exfoliation begins and the process terminates, sometimes leaving a temporary pigmentation.

The action of very strong irritants may result in necrosis of the skin and sometimes of deeper tissues. Detachment of the necrotised area leaves an ulcer which heals by forming a scar. Such changes are observed in third degree burns and frostbite, in burns with strong acids and alkalis, and in third degree roentgen dermatitides. If an irritant acts on the skin over a long period of time, a chronic dermatitis develops—the foci are purplish-reddish, the skin is thickened and hardened, and it exfoliates.

Chronic dermatitis differs from eczema mainly by its course; chronic dermatitis diminishes soon after the irritant has ceased to act and rather soon disappears altogether, whereas chronic eczema runs a much more stubborn course and easily relapses.

Artificial (simple) dermatitides are thus a normal (normergic) reaction to the effects of an unconditioned irritant.

Allergic dermatitis (Fig. 71) develops in response to the action of a relative (conditioned) irritant or allergen on the skin of persons who are hypersensitive to the given allergen (allergic or hyperergic reaction). The role of allergens may be played by various substances of vegetable and animal origin (pollen, sap, gum from bushes and trees, etc.), chemical substances (nickel and chromium salts, and synthetic tars), certain dyes (ursol, nitro-dyes) and medicaments (ammoniated mercury ointment, iodine tincture, solutions of corrosive mercuric chloride, novocain, streptomycin, etc.).

Unlike artificial dermatitis allergic dermatitis does not develop in all persons acted upon by a relative irritant. For allergic dermatitis to develop the patient must be extremely sensitive to the given irritant.

Allergic dermatitis usually develops, not at the first contact with the allergen, but some time later (most commonly from 5 days to 3 weeks later). During that time the reactivity of the organism is altered, an allergic state develops, and a repeated contact of the patient's skin with the allergen produces an inflammatory process in the irritated area. An important part in the development of hypersensitivity to external allergens is played by the internal state of the organism, the state of the nervous system, individual characteristics, working conditions, nutrition, etc. S. Pavlov has demonstrated by experiments on rabbits that they do not become sensitised to dinitrochlorbenzol if the allergen is applied to a denervated part of the skin. Endocrine disorders and diseases of internal organs are also of some importance.

At a factory, where there were cases of allergic dermatitis produced by nickel salts among the workers, we observed a woman

nickel-plater who had been well for 3 years. However, after a severe attack of influenza she developed allergic dermatitis. The attack of influenza weakened the patient's organism and facilitated her sensitisation to nickel. At another factory we observed workers developing allergic dermatitis due to chromium salts when excessively alkaline solutions began to be used in the shop. The constant contact of the workers' hands with strong alkaline solutions altered the normal acid reaction on the surface of the skin and made it more sensitive to various irritants.

The clinical picture of allergic dermatitis is similar to that of simple dermatitis and, especially, that of eczema. Redness and edema appear on the affected part, and the skin becomes hot to the touch. Intensification of the inflammatory phenomena produces nodules which may develop into vesicles or blisters. The vesicles and blisters burst and form erosive, exudative surfaces. The patient complains of itching and burning. As in simple dermatitis, the inflammatory process localises in the irritated area. However, the inflammatory process may subsequently also appear on other parts of the skin.
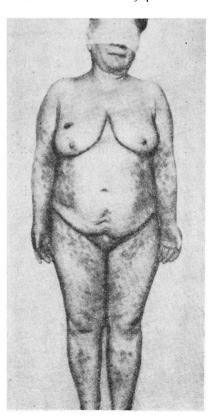
Fig. 72. Toxicoderma

When the irritant ceases to act the inflammatory phenomena begin to abate—the exudative areas dry, the redness pales and exfoliation begins. Allergic dermatitis heals more slowly than simple dermatitis. In patients with allergic dermatitis the increased sensitivity to the substance which plays the part of the allergen may be demonstrated by a cutaneous compress or drop test with this allergen (see Supplement, p.336).

Allergic dermatitis may develop into eczema when the patient's contact with the allergen continues. In cases of the patient's prolonged or repeated contact with the allergen the state of heightened sensitivity may increase. This may lead to "group allergy", i.e., the appearance of hypersensitivity in the patient not only to the given allergen, but also to closely related substances.
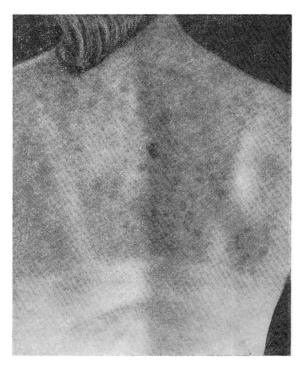
Fig. 73. Toxicoderma caused by administration of acrichine (quinacrine)

Toxicodermas (Fig. 72) are dermatitides in which the inflammatory process in the skin develops as a result of the action of an external irritant either introduced through the digestive or respiratory tracts, or administered subcutaneously, intramuscularly or intravenously. These irritants may be various foodstuffs, pollen, such substances as gas or dust, and certain medicaments administered per os or by injection.

In most cases toxicodermas are diffuse eruptions of a macular, nodular or vesicular character (Fig. 73). Eruptions of other lesions—pustules, blisters and nodes—are also observed. The eruptions are accompanied by itching and sometimes by pyrexia. Toxicodermas caused by consumption of certain foodstuffs are most commonly of a macular character, simulating a morbilliform eruption in some cases and a scarlatiniform eruption in others. They are caused by various foodstuffs, but most frequently by meat, fish, eggs, canned foods and highly seasoned dishes. Toxicodermas are often due to consumption of poor-quality or spoiled foodstuffs.

Fig. 75. Fixed sulfonamide erythema
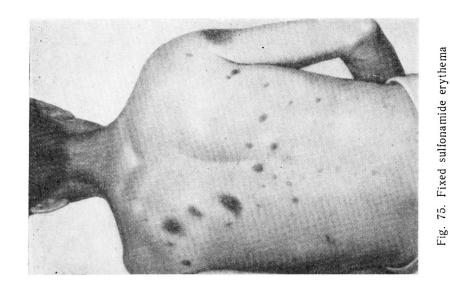
Fig. 74. Toxicoderma caused by injections of penicillin

The clinical pictures of toxicodermas caused by medicinal substances vary more widely. In addition to macular morbilliform and scarlatiniform eruptions there are also eruptions of pustules, blisters and nodes, as, for example, the eruptions due to intake of bromides and iodides.

Toxicodermas most commonly develop as a result of peroral, intravenous, intramuscular or subcutaneous administration of arsphenamine and sulfanilamide preparations, antibiotics, arsenic, iodides, bromides, aminopyrine, antipyrine and acrichine (Figs. 73 and 74). The antibiotics often producing eruptions are streptomycin and synthomycin (chloramphenicol).

So-called sulfonamide fixed erythema is not a very rare occurrence (L. Mashkilleison et al). In this form of toxicoderma round purplish or purplish-red, sharply defined macules from a few millimeters to several centimeters in diameter (Fig. 75) appear on various parts of the skin. The appearance of these macules is accompanied by itching and burning. Blisters the size of a plum are often formed on the macules. In many patients such macules and blisters simultaneously appear on the skin of the external genitals and on the oral mucosa. After bursting the blisters leave erosions which are particularly painful on the oral mucosa.

Treatment. The treatment of artificial dermatitides requires primary elimination of the irritant. If this requirement is satisfied, it is enough in most cases to administer local, indifferent treatment (lotions, suspensions, pastes) for all the symptoms of dermatitis soon to disappear. Cases with deep destruction of tissues—third degree burns and frostbite, chemical burns with strong acids and alkalis, and third degree roentgen dermatitis—are exceptions. They require general and long local treatment, best of all in a hospital.

The treatment of patients affected with allergic dermatitis must include, in addition to external agents, desensitising and antihistaminic agents.

The treatment of patients affected with toxicodermas must also begin with exclusion of the substances which caused the disease. Moreover, measures must be taken to eliminate the irritant from the organism as soon as possible. If the toxicoderma was caused by administration of medicines or ingestion of foodstuffs, a purgative is prescribed. To expedite the elimination of the irritant from the organism, the patient is also given plenty to drink and diuretics.

The other agents of general treatment administered intravenously or per os are calcium chloride, sodium hyposulfite, novocain, dimedrol (diphenhydramine), ephedrine and bromides.

Indifferent suspensions and pastes, and in cases of vesicles and erosions—lotions, are used in topical external treatment of patients affected with toxicodermas.

Occupational Skin Diseases

Occupational skin diseases arise in connection with the peculiarities of the patient's occupation. The development of occupational skin diseases depends on: (1) the character, strength and duration of the action of the irritant, (2) the general condition of the organism and its resistance to the harmful effects of the irritant, and (3) the sanitary and hygienic conditions of work.

The stronger and longer the action of the occupational irritant, the poorer the worker's health and the less the rules of hygiene and sanitation are observed at the place of work, the greater are the chances for developing occupational skin diseases.

Occupational diseases are divided into occupational signs and occupational skin diseases proper.

The occupational signs include such changes in the skin and its adnexa as calluses on the hands of manual workers, destruction of washerwomen's nails, etc.

The occupational skin diseases proper include various skin diseases caused by harmful influences of occupational conditions—high external temperature (in hot shops), constant humidity, dust and small solid particles (in textile plants, mines, flour mills, etc.), minor injuries (in metal working), products of oil refining and coal processing, acids, alkalis, salts, dyes, organic solvents and other chemical substances.

Development of occupational skin diseases is facilitated by various pathologic deviations in the worker's organism. Persons with chronic diseases of the nervous system and internal organs (diabetes, anemia, renal diseases, etc.) are more susceptible to occupational skin diseases. People with a tendency to hyperidrosis poorly tolerate work in hot shops where they readily develop prickly heat or dermatitis due to irritation of the skin by sweat. In cases of work with mineral oils occupational oil folliculitides occur more frequently in persons with abundant hair on the body and in those affected with seborrhea and acne.

The most common occupational skin diseases are occupational dermatitides and simple and allergic eczemas.

Occupational dermatitides are caused by various mechanical, thermal and, especially, chemical occupational irritants. The chemical substances with which workers often have to do in production are acids, alkalis, turpentine, organic solvents and other substances which may produce dermatitides. Diluted acids cause dermatitides, hyperkeratosis, fissures and ulcers. Diluted alkalis lead to defatting of the skin, its dryness, fissures and excessive sweating of the palms of the hands.

Organic solvents and turpentine defat and irritate the skin, make it dry, produce fissures in it and cause dermatitides.

Under production conditions occupational irritants usually act over a long period of time, and occupational dermatitides in many cases therefore become chronic. Very often repeated contact with the irritant (allergen) gradually leads to development of an allergic state.

Under production conditions occupational allergic dermatitis and occupational eczema may be caused by nickel, cobalt and chromium salts, certain dyes, synthetic tars and other substances. Occupational oil folliculitides are often observed in workers who have to do with mineral oils— products of petroleum refining and coal processing. Oil folliculitides frequently lead to development of furunculosis (Fig. 76).

Occupational pyodermas occur in connection with minor skin injuries in workers of the engineering, metallurgical, coal and peat mining industries. Workers of the fish-packing industry engaged in processing and salting
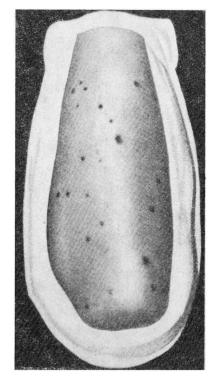
Fig. 76. Occupational oil folliculitis

fish sometimes develop ulcers on the fingers and hands due to irritation by the saline solution of the small wounds and abrasions caused by fish bones, fins or scales.

Building workers may have sustain burns during slacking lime. Ulcers with a scab in the centre surrounded by a red elevation of an inflammatory infiltrate ("bird's eye") are usally formed at the sites of the burns. Milder burns of the same type may also arise during preparation of mortar. During work with cement allergic dermatitides caused by the admixture of chromium salts may also be observed.

The *treatment* of occupational skin diseases must begin with elimination of the irritant. Many occupational skin diseases, including dermatitides, are quickly cured after the contact with the irritant has ceased. The general and topical treatment is conducted on the same principles as that of similar diseases of nonoccupational origin.

Prevention of occupational skin diseases is based on the following measures:

1. Sanitary engineering measures aimed at eliminating or reducing the harmful action of occupational irritants on the skin. For this purpose extensive mechanisation, automation and hermetisation of the production processes are introduced, special fixtures protecting the workers' skin from contact with irritating substances are made, and the technology of production is continuously improved.

2. Sanitary and hygienic measures—maintenance of cleanliness in the shops and at the working places, and provision of adequate ventilation and lighting.

3. Observance of the rules of personal hygiene by the workers—personal cleanliness, regular bathing and changing of underwear.

4. Wearing of working clothes to protect the workers' skin from contact with harmful substances. Regular changing, washing and repairing of the working clothes.

Protective ointments and pastes (IER-1, IER-2, KhIOT-6 [paste containing gelatin, starch, glycerin, Burov's solution and distilled water], etc.) are in some cases used to protect the exposed parts of the body from contact with irritating substances. Rubbed into the skin before work they create a film impermeable to the irritant on the surface of the skin. In many cases, however, this film is insufficiently strong, for which reason these ointments and pastes may be used only in a limited number of cases.

5. Cleansing the skin after work, i.e., thorough washing of the soiled parts under the tap, in a shower bath or bathtub.

Washing pastes—Rakhmanov's paste and Selissky's soap and lanolin paste—are successfully used to cleanse the skin. The pastes are rubbed into the skin after work and are then washed off with warm water. They cleanse the soiled skin and prevent it from becoming defatted.

6. Treatment of minor injuries with a 2 per cent iodine tincture or a 1 per cent alcohol solution of methyl violet, brilliant green, or Novikov's solution (antiseptic preparation containing brilliant green, ethyl alcohol and castor oil). It is at the same time necessary to instruct the workers as regards first self- and mutual-aid.

An important method of preventing occupational skin diseases is proper selection of workers for shops where there are such diseases. Workers who had eczema must not be assigned to shops where allergens are used, or workers with seborrhea or acne vulgaris to shops where mineral oils are used, or persons with a dry skin or hiperidrosis to hot shops.

Preventive measures against occupational skin diseases carried out in a planned manner have effected a considerable decrease in their incidence in the U.S.S.R.

ECZEMA (ATOPIC DERMATITIS)

Eczema or *atopic dermatitis* is one of the most widespread skin diseases.

Eczema patients constitute about one-fourth of all skin disease cases. The term eczema is derived from the Greek word *ekzein*, meaning "to boil over" because the numerous vesicles appearing in acute eczema somewhat resemble those forming in boiling water.

Eczema is an acute or chronic inflammatory skin disease involving the epidermis and the superior part of the derma. The disease is characterised by a widely varying clinical picture, itching and tendency to relapses.

Soviet scientists have found that eczema may be due to disturbances in the activity of the central nervous system.

P. Nikolsky has emphasised the connections between various functional disorders of the nervous system and development of eczema.

The works of I. Pavlov and his pupils (M. Petrova et al) have played an important part in elucidating the development (pathogenesis) of eczema. I. Pavlov observed different skin diseases, including eczema, developing in dogs after operations in the abdominal cavity, and proved the reflex origin of these skin eruptions. M. Petrova noted the appearance of eczema in dogs with experimental neuroses and explained it by a disturbance in the regulatory influence of the cerebral cortex.

Eczema is an *allergic* disease. Eczema patients exhibit increased sensitivity to various irritants.

The allergic essence of eczema must be understood as the result of repeated irritations of the receptors of the skin or the internal organs. The central nervous system responds to the new irritations of the receptors with pathologic reflexes which lead to development of eczema.

The role of the receptor irritants causing an allergic reorganisation of the organism may be played by various external and internal influences. The same chemical substances, medicaments and substances of vegetable and animal origin which cause allergic dermatitides may serve as external irritants or allergens in cases of eczema.

Eczema caused by contact with the primrose, an indoor plant, may serve as an example of this disease produced by external irritants. Some persons develop acute eczema on the hands and face

from a mere contact with the primrose. The increased sensitivity to the primrose may be so great that it is enough for a patient to stay a while in a room with this plant to have a new attack of eczema. Various chemical substances—certain dyes, turpentine, etc.—act as irritants causing an aggravation or new attack of eczema in many patients.

Substances which gain entrance into the organism through the digestive and respiratory tracts or through subcutaneous, intramuscular or intravenous administration may also act as allergens in eczema cases. Cases of relapses or aggravations of eczema have been known to be caused by consumption of lobsters, eggs and other foodstuffs, and administration of certain medicines per os.

A very important part in the development of eczema is also played by internal irritants forming in the organism in cases of helminthiasis, chronic constipation, diseases of the stomach, intestines, liver, endocrine glands and metabolism, and in chronic focal infections (chronic tonsillitides, appendicitis, adnexitis, sinusitis, carious teeth, etc.). The pathogenic substances formed in the organism of such patients may serve as *internal allergens* or *autoallergens*. If the case of eczema is associated with the presence of a focus of chronic infection in the patient's organism, for example, chronic tonsillitis, such a patient often exhibits a markedly positive reaction to intracutaneous administration of 0.1 ml of staphylococcus antigen (vaccine, anatoxin).

Eczema is rather frequently observed in diabetics. Women often have eczema in association with pregnancy, childbirth and menstruation.

As a rule, eczema patients exhibit increased sensitivity not to one, but simultaneously to several, and not infrequently to very many, allergens (polyvalent or nonspecific sensitisation).

The patient's sensitivity to various irritants often increases in the course of the disease. In some patients the number of substances which act as irritants continuously grows in association with the intensified allergic state. The substances which formerly did not irritate the skin become strong irritants and cause relapses and aggravations of eczema.

Eczema is sometimes called atopic dermatitis in order to emphasise the patient inborn tendency to development of an allergic state with respect to various external and internal irritants. Eczema occurs at any age—from infancy to senility—and may affect any part of the skin. Usually it affects the exposed parts of the body—face, hands and legs.

Acute and chronic eczemas are distinguished according to their clinical course.

In acute eczema the inflammatory process develops rapidly. The skin becomes red and edematous; subsequently red papules

Fig. 77. Acute eczema

the size of a pinhead or millet seed appear on the skin. The inflammatory process keeps progressing and the edema and formation of a serous exudate increase, for which reason the papules quite soon develop into vesicles. The vesicles are small, usually not larger than a millet seed, with a very thin cover. They soon burst and give rise to erosions. The erosions often coalesce into a continuous exudative surface (Fig. 77). The exudation soon diminishes and the exudate on the erosive surface dries into crusts. The crusts gradually detach themselves, the exfoliation on the affected part of the skin continuing for some time. The course of acute eczema may thus be divided into several successive stages. The development of redness corresponds to the *erythematous* stage and the formation of papules—to the *papular*. The period of transformation of papules into vesicles is called the *vesicular* stage, and the period of bursting of the vesicles and formation of an exudative erosive surface—the *exudative* stage. These are followed by the *crust formation* stage and *desquamative* stage. Addition of pyogenic infection leads to formation of *pustules* and *purulent crusts* (Fig. 78). Acute eczema is characterised by a rapid succession of the stages and intense subjective sensations—tension, burning and, especially, itching. The itching is often so intense that it forces the patients to scratch their skin and disturbs their rest and sleep.
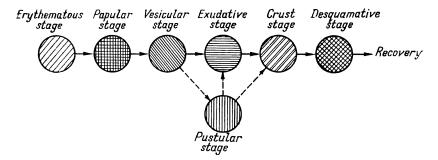
Fig. 78. Diagram showing development of acute eczema

A characteristic peculiarity of acute eczema is the polymorphism of its eruption, i.e., the simultaneous existence of various lesions—erythema, papules, vesicles, erosions, crusts and scales— on the skin. Since all these lesions are but different stages of development of acute eczema this polymorphism is called *pseudopolymorphism*. However, all the aforementioned stages are not always observed in acute eczema. The process may stop at any stage and be resolved. For example, the vesicles may by-pass the exudative stage, dry and change to crusts. The papules do not always change to vesicles, but sometimes gradually pale, become flat and begin to scale. Similarly papules do not always form in the erythematous areas. Consequently, both pseudo- and true polymorphism are observed in acute eczema.

The foci of acute eczema are not clearly defined, the inflamed part of the skin shading off into the surrounding unaffected parts.

The symmetrical arrangement of the foci of eczema is very typical, i.e., in most cases of both acute and chronic eczema the foci of the disease arrange themselves on both sides of the body.

In most cases acute eczema lasts several weeks and either terminates in healing or develops into the chronic form. In many cases of acute eczema the cure is not stable and after a while the disease recurs, i.e., it relapses. Recurrent eczema gradually assumes all the features of chronic eczema.

In chronic eczema there is no bright redness, acute edema, clearly defined polymorphism or rapid succession of stages. Phenomena of cellular infiltration in the papillary layer of the derma come to the foreground. The skin thickens and becomes hard to the touch, the cutaneous pattern coarsens, and hyperkeratosis and exfoliation develop (Figs. 79 and 80). The main subjective sensation of chronic eczema patients is itching which may be very intense.

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Chronic eczema may last an indefinitely long time — years and even decades. A cyclic course of the disease is usually observed — periods of improvement and even apparent cure alternate with aggravations and relapses. The duration of the remissions or disappearance of the eruptions (lucid intervals) and periods of aggravation or relapses vary in different patients. The differences are due to different conditions in which the patients find themselves, individual characteristics of each patient's organism, and, of course, the quality and regularity of the treatment.

In addition to ordinary eczema (acute and chronic), several other clinical varieties of this disease are distinguished.

Seborrheic eczema is a frequently occurring variety of eczema. In this form of the disease the process is most commonly localised on the scalp, on the face, in the folds behind the ears, on the neck, in
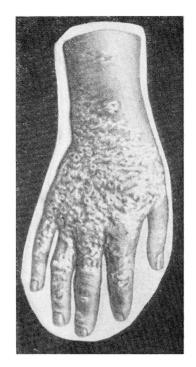
Fig. 79. Chronic eczema

the interscapular region, on the sternum, in the popliteal spaces, and in the region of the navel and pubes. Seborrheic eczema most frequently begins on the face or on the scalp. At first reddish and rather clearly defined macules with a yellowish hue appear. The skin in these areas is moderately infiltrated. The macules are covered with oily, yellowish scales. Vesicles form infrequently and therefore there is usually no exudation. The latter is observed only when seborrheic eczema is localised in skin folds—inguinal, axillary, behind the ears, under women's breasts, and on the scrotum. The macules, at first small—the size of a finger-nail or a small coin—gradually enlarge. They often coalesce, forming irregular figures with peculiar outlines. Seborrheic eczema is accompanied by itching of varying intensity.

Microbial eczema is characterised by clearly defined foci of affection, saturated red colouring and development in most cases around a wound, ulcer or pustule. An important part in the development of this variety of eczema is played by pyogenic microbes. On coming in contact with the skin from a pustule, infected wound, chronic ulcer or a fistula these microbes and their waste products cause prolonged irritation of the given part of the skin.

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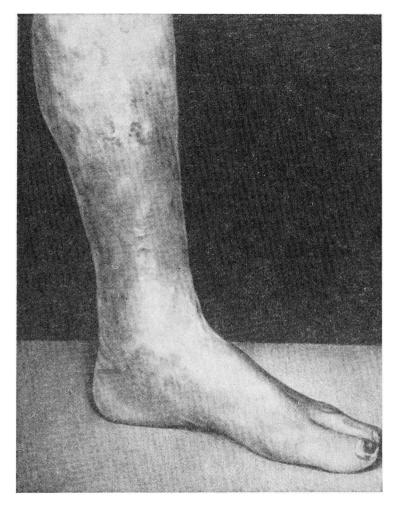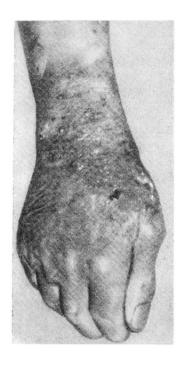
Fig. 80. Chronic eczema of the shank associated with varicose veins
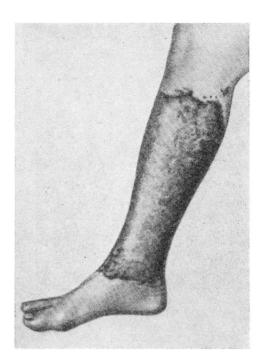
Fig. 81. Paratraumatic
eczema

Fig. 82. Microbial eczema

Increased sensitivity to these irritants gradually develops and manifests itself in formation of a focus of eczema at the site of irritation.

Microbial eczema often develops around wounds sustained in war and about other injuries, in which cases it is called *paratraumatic eczema* (Fig. 81). The development of this disease is favoured by a copious purulent discharge from a wound, its unclean maintenance and the use of irritating medicaments. Microbial eczema is often a complication of chronic ulcers of the shank, for example, in cases of varicose veins. It also develops around long unhealing pustular eruptions—furuncles and ecthyma—and various fistulas.

Microbial eczema is in most cases unilateral, unsymmetrical.

A border of desquamating epidermis is often observed along the edges of the foci of microbial eczema. The borders of the foci may have scalloped outlines; their central part becomes saturated red and is covered with lamellar scales or purulent crusts. In aggravated cases exudation develops all over the surface of the focus or over part of it (Fig. 82).

Fig. 83. Exudative eczema in child (from A. Kartamyshev)

Infantile eczema most commonly develops in nurslings and infants in connection with gastrointestinal dysfunction. In such cases regurgitation, diarrhea, vomiting and constipation are observed. The development of eczema is often associated with disturbances in nursing, especially with too plentiful and frequent nursing. Infants affected with eczema are often overnourished and have loose ("pasty") subcutaneous tissue. They often exhibit symptoms of so-called exudative diathesis—a tendency to upper respiratory catarrhs, diseases of the middle, ear and chafing in skin folds. In infants 1-2 years of age aggravations and relapses of eczema are often associated with helminthiasis and consumption of certain food-stuffs—eggs, cow's milk, sweets and farinaceous foods. After consumption of these foodstuffs the eczema either recurs or becomes aggravated.

In children past 2 years of age eczema is not infrequently due to increased sensitivity to various external irritants. These irritants may be woolen clothing, the fur of cats and dogs, dandruff, pollen and other substances.

The face and the hairy part of the head are the favourite sites of eczema in children. A redness and itching appear on the face, in the region of the cheeks and forehead and are soon followed by small papules and vesicles. Then the vesicles burst and a bright-red, exudative surface is formed (Fig. 83). The exudate dries and leaves the face covered with crusts. A secondary infection often develops—impetiginous pustules appear and the exudative areas become covered with heavy yellow-grey crusts. The intense itching makes the child scratch the affected areas till they bleed; the child becomes restless, often cries, sleeps badly and loses his appetite.

In many children eczema spreads to the neck, trunk and limbs. At first small foci of redness and edema appear in these areas and become covered with papules and vesicles. Soon the vesicles burst and exudation develops. Since new lesions continuously appear on the periphery of the foci the latter enlarge and may coalesce.

Infantile eczema is a protracted and stubborn disease which subsides and even disappears, but then reappears. The disease may persist until the age of 2-3 years, after which it usually passes off. In some children, however, it does not pass off even at this age and assumes the character of usual chronic eczema.

In some cases eczema is complicated by secondary infection—pyoderma—which is favoured by scratching and the presence of an exudative surface that facilitates implantation of microbes in the skin.

In disseminated acute or chronic aggravated eczema the inflammatory process may sometimes involve the entire skin. This is observed in progressive eczema when new eczematous foci continuously arise and the existing ones gradually enlarge and coalesce. This condition is called *secondary erythroderma*. In these cases the skin is bright-red, edematous and infiltrated; in some areas it presents an exudative surface, in others it is covered with lamellar scales. Erythroderma patients are restless and irritable; they complain of intense itching, constant chills and a sense of tension in the skin. Their body temperature is usually elevated to 38°C.

Eczema must be distinguished from artificial and allergic dermatitis. In simple dermatitis the patient is not hypersensitive to various external irritants observed in eczema patients. The inflammatory process is caused by a strong "unconditioned" irritant. Dermatitis develops only on the part of the skin acted upon by the irritant. As soon as the action of the irritant ceases the inflammatory process begins to subside. Artificial dermatitis relapses only when the skin is again acted upon by this irritant.

Eczema differs in that inflammatory foci may appear after external irritating influences, as well as without them. Not infrequently the inflammatory process in the eczematous foci persists for a long time despite the discontinued action of the irritant. In eczema patients new foci often appear on remote parts of the skin which were not acted upon by the irritant.

It is more difficult to distinguish eczema from allergic dermatitis. In allergic dermatitis the patients are also hypersensitive to the external allergen, most commonly to one, but sometimes to several substances closely interrelated in their properties, whereas in eczema hypersensitivity develops to very many external irritants differing in their character and properties.

In allergic dermatitis patients the inflammatory process may extend beyond the area acted upon by the irritant. New foci in areas far removed from this part of the skin may also form. However, after contact with the allergen has ceased the process gradually subsides and no relapses without new contact with this allergen are observed. The symptom characteristic of eczema—pinpoint exudation is not so clearly marked in allergic dermatitis and does not persist so stubbornly as in eczema.

In some cases allergic dermatitis may develop into eczema.

Treatment. Treatment of eczema is always a complicated, although soluble problem, if the patient is not treated in a routine manner and the factors causing and bolstering up the disease are analysed.

The patient must be thoroughly examined before treatment. It is sometimes necessary to consult other medical specialists — neuropathologists, internists, gynecologists, etc.

It is necessary to eliminate all unfavourable influences on the patient's nervous system and establish an appropriate regimen — long hours outdoors, adequate rest and sleep, physical exercise and sports (in accordance with the patient's age and general condition). The patient with acute eczema is prescribed a vegetable and dairy diet with a lot of vitamins and a limited amount of salt. Mainly boiled meat is recommended. Seasoned foods and spices are prohibited. Foodstuffs causing aggravation of the eczematous process and alcoholic beverages must be excluded from the diet of chronic eczema patients.

The diet of children affected with eczema requires special attention. These children must not be overfed; they must be fed strictly by the clock. To find the foodstuffs which cause eczema, an elimination test diet is prescribed, i.e., a diet in turns excluding eggs, cow's milk, etc.

No less important a task in the treatment of concurrent diseases of the eczema patient. The course of eczema very often improves after a cure or improvement of the patient's internal disease, expulsion of intestinal worms, and establishment of daily bowel movements in cases of chronic constipation.

Sometimes it is possible to find the substance which serves as the allergen for the given patient and to discontinue the patient's contact with this allergen. In such cases the treatment of eczema is greatly facilitated and sometimes it passes off even without treatment.

Moreover, for the *general treatment* of eczema patients the following methods and agents are resorted to: methods of influencing the nervous system (novocain intravenously, per os and in the form of a block, vitamin B_1, sodium bromide, neuroplegic and ganglion blocking substances), desensitising methods (calcium chloride, sodium hyposulfite, ascorbic acid, etc.), endocrine preparations, vitamin therapy (nicotinic acid, riboflavin, vitamin B_6, vitamins C and A), and antihistaminic preparations. Some patients, especially those with seborrheic eczema and microbial eczema, greatly benefit by autohemotherapy and injections of laky blood.

Eczema in children is effectively treated by injections of maternal blood. In progressive disseminated eczema and in eczematous erythroderma excellent results are not infrequently produced

by blood or plasma transfusions and by administration of corti-costeroid hormones.

Local, external treatment of eczema must be administered according to the stage of the disease and the sensitivity of the individual patient's skin. In acute eczema, in the stage of vesicle formation and exudation lotions and moist-desiccant dressings with indifferent solutions (lead water, 0.25 per cent silver nitrate, etc.) are used. As the acute inflammatory process abates, applications of oil suspensions (zinc oil), water suspensions and pastes (Lassar's paste, 1-2 per cent ichthyol paste, etc.) are resorted to. In cases of infiltration of the skin pastes and ointments containing naphthalan oil (5-50 per cent) and pitch (1-10 per cent) are prescribed after abatement of the acute inflammatory phenomena. The concentration of these substances in the pastes and ointments is gradually increased with the patient's habituation to them. Good antipruritic and anti-inflammatory effects are produced by ointments containing corticosteroid hormones which can be prescribed in combination with pitch preparations (see Supplement, p. 327). Resorption of the infiltrate is facilitated by applications of paraffin or ozocerite.

As for physiotherapeutic methods, good effects are produced by Bucky's rays. Roentgen rays are also used in the treatment of eczema. However, caution must be exercised in prescribing roentgen treatment. Roentgen therapy really favours resorption of the infiltrate in the foci of chronic eczema, but roentgen rays are not indifferent to the organism and do not prevent relapses of eczema.

Roentgen rays are therefore used mainly in circumscribed foci of chronic eczema with marked infiltration of the skin if other forms of general and local treatment fail to produce an appreciable effect.

The skin of eczema patients does not tolerate water, for which reason patients with acute eczema are prohibited from washing the affected areas. Instead of washing they are advised to cleanse the soiled skin with vegetable or vaseline oil.

At the same time prolonged soiling of the skin may lead to its irritation. Chronic eczema patients are therefore allowed to wash once a week with warm water and children's or lanolin soap, but lathering and sponging the affected areas are prohibited.

Occupational eczema. Among the external factors producing eczema there are some which are connected with the work done by the patient and with the working conditions. For example, some workers in hot shops develop eczema in connection with the high external temperature and constant irritation of the skin by sweat.

Occupational eczema is particularly frequently caused by various chemical substances. Workers of fur-dressing factories

engaged in dyeing furs develop eczema of the hands and face because of their contact with ursol (a fur dye). Work with certain artificial tars may give rise to acute eczema of the face, hands and scrotum. Nickel-plating of metal parts causes in some workers eczema of the hands and face with very intense itching—"nickel-plater's eczema".

Transfer of the worker who has contracted occupational eczema to another occupation usually puts an end to the disease, but if the worker stays at his former job the disease often assumes a stubborn course and spreads to other areas of the skin. In such cases even transfer to another occupation may not bring any particular relief.

Occupational eczema is also observed in medical workers, for example, in nurses making injections of streptomycin, if the solution comes in contact with their hands during the injections or during the washing of the syringe. Dentists may develop eczema of the hands if the novocain solution comes in contact with their hands. Surgeons, surgical nurses, midwives and other medical workers sometimes develop eczema as a result of frequent irritation of their hands when scrubbing them with disinfectants. In all such cases it is a matter of hypersensitivity of certain persons to the aforementioned substances.

Prophylaxis of occupational eczema consists in assanation of the working conditions. The more perfect the technology of production and the greater the efficiency, the lower the incidence of occupational eczema among the workers.

Proper ventilation of the working premises, wearing work cloths, application of protective ointments to the hands and face, and obligatory showering after work are the most important measures for preventing occupational eczema.

PRURITIC SKIN DISEASES

Neurodermatitis

Neurodermatitis is a disease that has a good deal in common with eczema. Dysfunction of the central nervous system plays a very important part in the origin and course of the disease. Neurodermatitis is called neurosis of the skin manifested in intense itching. It is favoured by gastrointestinal diseases, endocrine disorders, hemorrhoids and chronic intoxications (alcoholism, etc.). There are two forms of neurodermatitis—circumscribed and disseminated.

Circumscribed neurodermatitis begins with the appearance of intense itching which is paroxysmal and greatly discomforts the patient, especially in the evening and at night. The itching disturbs the patient's sleep. Very often patients scratch their skin till it bleeds, which somewhat relieves the itching.

Itching is for a rather long time the only symptom, eruptions on the skin in the form of small, somewhat shiny, reddish flat papules appearing later. The papules gradually coalesce forming infiltrated patches in the centre of which the skin is thickened, hardened, has a coarse pattern and is covered with lamellar scales. Separate, flat, somewhat shiny papules can be seen on the periphery of the patches. Yellowish-brownish pigmentation is observed still further on the periphery. The foci of neurodermatitis are often covered with sanguineous crusts formed as a result of scratching.

In patients with circumscribed neurodermatitis the patches are quite clearly defined, but are not very many (Fig. 84). They are most commonly localised on the posterior surface of the neck, in the elbow bends and popliteal spaces, often symmetrically. Unlike eczema, vesicles do not form on the foci of neurodermatitis, and exudation develops very rarely. The disease runs a protracted course, remissions alternating with aggravations. Owing to scratching, neurodermatitis is sometimes complicated by eczematisation (redness, edema, some exudation) and secondary pyoderma.

Neurodermatitis usually occurs between 20 and 50 years of age, more often in women than in men.

Diffuse neurodermatitis occurs less frequently and is a severe disease with a stubborn course. It begins with attacks of tormenting itching. Extensive portions of the skin become thickened, dry, and, as a result of scratching, covered with scales and sanguineous crusts. The skin pattern becomes coarse and the colour dirtyish with yellowish or brownish pigmentation. Diffuse neurodermatitis is very closely related to certain disseminated forms of eczema (atopic dermatitis).

Treatment. In instituting treatment of neurodermatitis patients it is necessary, as in the treatment of eczema, to examine the patient and take measures to eliminate the discovered infractions of the patient's regimen and to treat the concurrent diseases of internal organs and the nervous system.
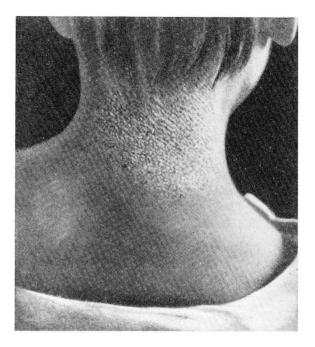
Fig. 84. Circumscribed neurodermatitis (from A. Zenin and N. Torsuyev)

Good effects in the treatment of circumscribed neurodermatitis are produced by administration of aminazine (chlorpromazine), nicotinic acid, novocain, bromides, and hypnosis. Agents causing resorption of the infiltrate are used locally, namely, applications of paraffin, ozocerite, pastes and ointments with naphthalan oil (10-50 per cent) and pitch (2-10 per cent), as well as ointments containing corticosteroid hormones.

Pruritus

Pruritus may be observed not only as one of the symptoms of eczema, neurodermatitis and other skin diseases, but also as an independent disease entity. In these cases examination of the skin shows it to be unaffected.

Pruritus may be circumscribed and general. Circumscribed pruritus particularly frequently affects the region of the anus and the external genitalia.

The itching may become unbearable and may force the patients to scratch the body. Scratching in cases of protracted and intense itching results in excoriations and lichenisation. Sometimes secondary pyoderma develops.

Pruritus is often paroxysmal.

Disturbances in the functions of the central nervous system play a very important part in the development of pruritus. Cases of development of pruritus after distressing experiences, trouble, etc., are very well known. Some patients develop pruritus at the mere sight of such parasites as lice, bedbugs and flies. There was a case of general pruritus in a woman who 8 months prior to that had scabies and was cured by inunction of a sulfur and pitch ointment. The mere sight and odour of sulfur and pitch ointment which was prescribed for her son caused in this woman a relapse of pruritus. In this case pruritus was of the character of a conditioned reflex stimulated by the sight and odour of the ointment.

Pruritus may be caused by chronic intoxications, metabolic disturbances, diseases of the internal organs, for example, pruritus senilis, pruritus of pregnancy, pruritus in liver diseases, in some forms of jaundice, helminthiasis and chronic constipation. Circumscribed pruritus ani and pruritus of the external genitalia is often associated with helminthiasis (pin worms, etc.), hemorrhoids, prostatitis, diseases of the female genitalia, and the climacteric.

The *treatment* of pruritus must be based on a careful examination of the patient to elucidate and eliminate the causes of the disease. The same general methods of treatment are used as for neurodermatitis. Dimedrol in a dose of 0.03 g 3 times per day or 0.05 g twice a day is helpful. Applications of alcohol solutions of carbolic acid, menthol and thymol, and ointments containing 2-5-10 per cent anesthesin or 2-3 per cent dimedrol, as well as rubdowns of the skin with vinegar, are used for external treatment. Especially effective in the treatment of pruritus ani and pruritus of the genitalia are corticosteroid ointments.

Urticaria

The urticarial eruption consists of one type of morphological lesions—wheals which may be red, pink or white, according to the extent of the edema. They vary in size from that of a hemp seed to that of the palm and larger, the latter resulting from coalescence of several wheals.

Wheals may be localised on any part of the skin. Their eruption is always accompanied by intense itching and a sense of burning.

Urticarial wheals do not last long—usually not longer than a few hours. However, new wheals may erupt over a period of a number of days and even weeks, and in chronic urticaria—over months and even years. Despite the intense itching urticaria patients do not scratch so much, and crusts, eczematisation and secondary pyoderma are therefore rarely observed.

Fig. 85. Urticaria. Eruptions at the site of mechanical irritation

The essence of this disease consists in hypersensitivity to various external and internal factors. Of the external irritants wheals are most commonly produced at the sites of irritation by insect bites (mosquitoes, midges, fleas, etc.) and contact with certain plants (nettle, etc.). Urticaria may also be produced by thermal influences, most frequently by the effects of cold. In some cases urticaria is caused even by slight mechanical irritation. Ciphers or letters may be inscribed with the blunt edge of a stick on the skin of such patients; in a few minutes these ciphers or letters will appear as wheals (Fig. 85). Various foodstuffs (strawberries,

spices, lobsters, etc.) and medicaments may also act as irritants. Urticaria may also result from intoxication in helminthiasis, chronic constipation, etc.

A very important part in the development of urticaria is played by dysfunction of the nervous system. Urticaria may be evoked by suggestion in a state of hypnosis. It can be cured by similar suggestion in patients hypersensitive to certain medicaments or foodstuffs.

Urticaria may be acute, i.e., it may develop suddenly and disappear quite soon, when the irritant which caused it ceases to act.

Chronic urticaria is characterised by repeated wheal eruptions. In the intervals between the relapses the skin may long have no eruptions at all. Chronic urticaria is usually associated with chronic diseases of internal organs of the nervous system, or the existence of a chronic infectious focus in the patient's organism (chronic angina, sinusitis, appendicitis, etc.).

Treatment. A purgative—Epsom or Glauber's salt—is prescribed and a vegetable and dairy diet with a limited amount of sodium chloride is recommended in cases of acute urticaria. Antihistaminic preparations—dimedrol and ephedrine—and desensitising agents—calcium chloride and sodium hyposulfite—are administered.

In chronic urticaria it is the most important to elucidate the cause of the disease and to eliminate or weaken it. Some chronic urticaria patients benefit from taking calcium chloride, sodium hyposulfite, adrenalin, dimedrol, ephedrine and bromides.

Topical, external treatment of urticaria consists in application of substances relieving the itching, namely, alcohol solutions of menthol (1-2 per cent), thymol (1-1.5 per cent), carbolic acid (1-2 per cent) and vinegar.

Strophulus Pruriginosus or Urticaria Papulosa

This disease is most commonly caused by various digestive disturbances—constipation, helminthiasis, improper diet (too plentiful or too frequent feeding) and intolerance of certain foodstuffs. The latter are most frequently eggs (especially the white of chicken eggs), certain sweets (candy, chocolate, pastries, honey), farinaceous foods and cow's milk. A considerable part in the development of this disease may be played by the child's hypersensitivity to insect bites (especially bedbugs, fleas, mosquitoes, sandflies). Strophulus usually begins in the first year of life. Wheals and rather hard conical papules the size of a hemp or millet seed appear on various parts of the skin. The papules are frequently surmounted by a small vesicle. The wheals and, especially, the papules cause intense itching which makes the patients scratch.

As it urticaria, the wheals of strophulus for the most part soon disappear without leaving a trace. The papules are more stable lesions and disappear only within 4-10 days. While old lesions disappear new ones keep appearing over a period of several weeks and even months. Then the eruptions disappear and some time later reappear.

Strophulus usually lasts until 2-3 years of age. The scratching caused by intense itching often leads to complication of strophulus by secondary pyoderma.

The *treatment* of the strophulus patient must be aimed primarily at regulating the patient's diet and re-establishing normal gastrointestinal activity. Eggs and sweets (candy, chocolate, pastries and honey), seasoned and canned foods are excluded from the diet, and consumption of farinaceous foods and cow's milk is limited (the latter too no more than 0.5 litre a day).

If the patient suffers from constipation or helminthiasis, it is necessary to strive for a daily stool and expulsion of the worms. The treatment also makes use of calcium chloride, sodium hyposulfite and injections of maternal blood. Local treatment consists in application of ointments containing corticosteroid hormones (1 per cent hydrocortisone, 0.5 per cent prednisolone, etc.), suspensions and alcohol solutions which mitigate the itching. Good effects are produced by bran, pine needle extract and potassium permanganate baths. A sine qua non for successful treatment of strophulus is extermination in the dwelling of all bedbugs and fleas by means of DDT and protection of the child against bites of sandflies and other insects with the aid of repellents (dimethyl phthalate) and other measures (protective nets, etc.).

Prurigo

Prurigo is a chronic itching skin disease of scarcely known etiology. It is believed to be caused by disturbances in the function of the central nervous system and chronic intoxications. P. Nikolsky was one of the first to note the importance of dysfunction of the nervous system in the development of this disease. He observed a marked white dermographism in many prurigo patients. As a rule prurigo begins in early childhood. In many patients the disease at first runs the course of usual pruritus or chronic urticaria, but at 3-4 years of age the picture of the disease changes. The number of compact itching papules continuously increases and the papules are localised mainly on the extensor surfaces of the extremities. Sometimes prurigo assumes its own characteristic picture from the very outset.

Intolerable itching forces the patient to scratch his skin till it bleeds, owing to which numerous sanguineous crusts and scars can always be seen at the sites of extensive scratching. There are

fewer papules on the trunk, face and flexor surfaces of the extremities. The papules are compact, conical, and scarcely elevated above the skin; they vary in size from that of a pinhead to that of a hemp seed. A very small vesicle may sometimes be seen on top of the papule. In the affected areas the skin is thickened, pigmented and has a coarse pattern (Fig. 86).
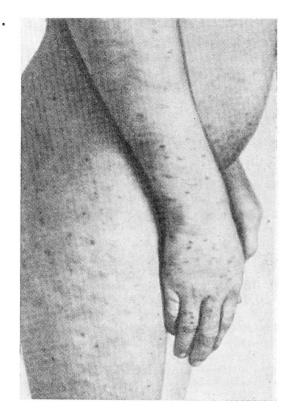
Fig. 86. Prurigo

The lymph nodes, especially the femoral, of prurigo patients are very frequently enlarged.

With a comparatively mild course the disease terminates between the 7th and 10th years of life or at the time of sexual maturation. Periods of remission alternate with periods of aggravation. In severe cases prurigo lasts many years and even all through the patient's life. In such cases the patient's general condition is seriously affected; the patient becomes irritable, and reticent, and owing to intense itching suffers from insomnia. As a result

of continuous scratching, the disease is quite often accompanied by eczematisation, lichenisation and secondary pyoderma.

Prurigo most commonly develops in anemic and emaciated children living in poor hygienic and material conditions ("disease of basements"). Great importance in the development of prurigo is, in particular, attached to the patients' hypersensitivity to bites of bedbugs, fleas and other blood-sucking insects.

Treatment. Prurigo patients must be ensured a proper regimen, adequate sleep, regular hours outdoors and an appropriate diet. They are prescribed vegetable and dairy foods containing a lot of fresh vegetables and fruit, sour milk and kefir. Spices, seasoned and salty foods are prohibited, and consumption of meat, eggs and sweets is restricted.

In cases of internal diseases and helminthiasis the patients are given corresponding treatment. The bedbugs and fleas in the patients' dwellings must be exterminated by means of DDT, and the patients must be protected against bites of other blood-sucking insects.

Good effects in the treatment of prurigo are produced by intravenous or peroral administration of novocain, calcium chloride, sodium hyposulfite, sodium bromide.

General roborants are also prescribed, namely, iron and arsenic preparations, phytin, fish liver oil, campolon, nicotinic acid, ascorbic acid, vitamins A and B_{12}, endocrine preparations and autohemotherapy.

Corticosteroid ointments and agents mitigating itching are used for local treatment; these are alcohol solutions of carbolic acid (1-2 per cent), menthol (1-2 per cent) and pitch (5-10 per cent). Favourable effects are produced by pitch baths.

In cases of lichenisation applications of paraffin, ozocerite, pastes and ointments containing pitch (2-10-20 per cent) or naphthalan oil (10-50 per cent) are prescribed.

Very good results are produced by health resort treatment (hydrogen sulfide baths, sea bathing and pelotherapy).

The patient's return to his usual conditions of life after successful treatment is often followed by a relapse of the disease.

PSORIASIS

Psoriasis is a chronic skin disease characterised by eruptions of superficial papules and larger patches with phenomena of chronic inflammation. It is a commonly occurring condition and constitutes 2-4 per cent of all cases of skin diseases. It often begins in adolescence and youth. Its incidence among men and women is about the same.

The *etiology* of psoriasis is unknown. Various investigators have suggested many theories to explain the origin of the disease. The theories of nervous and infectious origin of the disease have been given the best substantiation.

According to the theory of nervous origin, psoriasis is caused by disturbances in the functions of the central nervous system. A. Polotebnov and P. Nikolsky have established that psoriasis involves neurovascular disorders and disturbances in the secretion of sebum and perspiration. It is also very well known that psoriasis or its relapses sometimes develop after psychic traumas or "mental stress"; such facts were observed by A. Polotebnov.

The symmetry of the eruptions very often observed in these patients also denotes a disturbance in nervous activity of psoriasis patients.

A. Ukhin and A. Krichevsky have established a number of interesting facts denoting the viral origin of psoriasis.

However, the viral nature of psoriasis cannot be considered demonstrated and the etiology of this disease remains unknown. The theories explaining the origin of psoriasis by disturbances in metabolism, dysfunction of the endocrine glands and diseases of the internal organs are less substantiated.

The metabolic disorders, dysfunction of endocrine glands, etc., observed in some psoriasis patients may be the result and not the cause of psoriasis.

The principal lesion in psoriasis is a pink-red or red, flat, superficial papule slightly elevated above the level of the surrounding skin. Long existing papules, especially on the lower limbs, have a purplish hue. The papules soon become covered with whitish, often silvery-white lamellar scales. On scraping of a papule the number of scales increases and its surface assumes a resemblance to a drop of congealed stearin (stearin macule symptom). If all the scales are removed by scraping, the surface of the papule is found to be covered with a thin semitransparent film (psoriatic film symptom). Removal of this film reveals a saturated red

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surface of the papule with minute drops of blood (petechial or capillary hemorrhage symptom).

Psoriatic papules are distinguished for their regular round shape, clearly defined borders and tendency to enlargement. In the beginning they are not larger than a pinhead, but then gradually attain the size of a drop or a small coin. Upon enlarging the

Fig. 87. Psoriasis

Fig. 88. Psoriasis

adjacent papules coalesce and form pink-red infiltrated patches covered with silvery-white scales. These patches are usually scalloped, which indicates their formation by coalescence of individual papules (Fig. 87).

In cases where small papules the size of a pinhead prevail on the patient's skin the disease is called *punctate psoriasis*. If most of the lesions are the size of a drop or coin, the disease is referred to as *discoid* or *nummular psoriasis*. In cases where most of the papules coalesce into patches the disease is called *patchy* or *diffuse psoriasis* (Fig. 88).

Sometimes the psoriatic papules and patches gradually turn pale and flatten in the centre while their peripheral parts continue
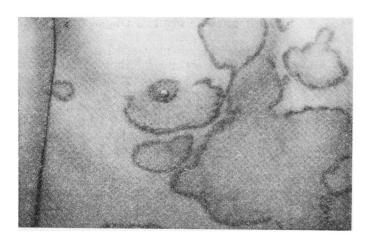
Fig. 89. Orbicular psoriasis
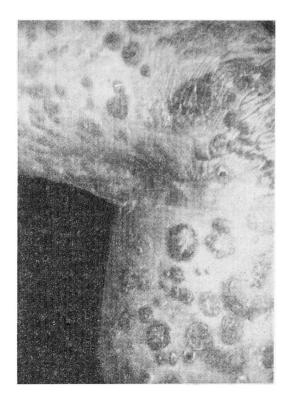
Fig. 90. Psoriasis

to enlarge. As a result of these changes ring-shaped papules and patches are formed—*orbicular psoriasis* (Fig. 89).

Psoriatic papules and patches may arise on any part of the skin, but they are localised mainly on the extensor surfaces of the limbs, especially the elbows and knees (Fig. 90). The hairy part of the head is also very frequently affected.

The eruptions of psoriasis are for the most part symmetrical. The psoriatic papules and patches are often so numerous that they cover the greater part of the skin. The eruptions appear comparatively rarely on the face and very rarely on the mucous membranes. Affection of the nails is observed in about one-tenth of the psoriasis patients. Punctate indentations form on the nails and their surface look like that of a thimble. In other cases the nails thicken, become uneven, lustreless and clawlike. Other forms of ungual dystrophy are also observed.

Most psoriasis patients have no subjective sensations or the latter are manifested in slight itching. In some cases the itching may be rather intense.

In course of time (different periods in different patients) the psoriatic papules and patches gradually turn pale, assume a purplish or brownish hue, become increasingly flatter, the number of scales on them decreases and, lastly, they are resorbed without leaving a trace. The psoriatic papules and patches disappear leaving a temporary pigmentation, more rarely a depigmentation.

Psoriasis is a chronic disease. However, its course varies widely. In some patients psoriasis involves numerous eruptions and frequent exacerbations and relapses. Such patients have few and short lucid intervals or remissions, i.e., periods when the skin is free of eruptions. In other cases psoriasis runs a more favourable course: there are few psoriatic lesions which are sometimes localised only on the elbows and knees, exacerbations and relapses are rare, while the lucid intervals last for years and even decades. These differences in the course of the disease depend on the general condition and reactivity of the patient's organism, the environmental conditions under which the patient lives and works, and the treatment.

Three stages are distinguished in the development of the psoriatic eruption.

The first or *progressive* stage is characterised by appearance of increasingly new lesions on the patient's skin and enlargement of the existing ones. At this stage, in addition to large papules and patches, the patients also have very small recently appeared papules.

Whitish scales do not cover the entire surface of the papules; on their periphery there is a narrow pink-red areola free of scales. This areola is a sign of the peripheral growth of the papules. In

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Fig. 92. Eruption of psoriatic papules at the site of pressure exerted by clothing (isomorphous reaction)
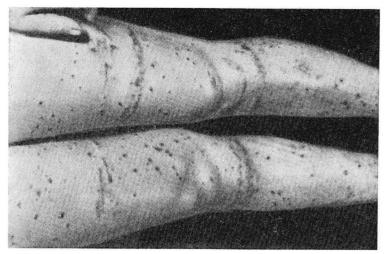
Fig. 91. Linear arrangement of papules at the site of mechanical irritation in psoriasis patient (isomorphous reaction)

Fig. 93. Psoriatic papules at the site of a tattoo (isomorphous reaction)

the progressive stage the patients rather frequently experience itching, usually moderate.

During the second stage—*stationary*—eruption of new lesions ceases. The extant papules and patches do not change in size and their surface is completely covered with scales. Of course, development of new papules and enlargement of various lesions are also possible in the stationary stage, but these phenomena no longer predominate in the picture of the disease as they do in the progressive stage. In the stationary stage the eruptive lesions may long persist without any appreciable changes.

The third—*regressive* stage is the stage of gradual disappearance, resorption of the psoriatic lesions. The papules and patches turn pale, assume a brownish or purplish hue, their infiltrate is resorbed and the number of scales on their surface gradually decreases. Lastly all lesions or only some of them disappear.

Elucidation of the stage of the disease in each individual case is of great practical importance for establishing a general regimen for the patient and prescribing the treatment. Special attention must be devoted to patients with the progressive stage of psoriasis when the most clearly marked changes in the reactivity of the organism are observed. These changes are, in particular, manifested in the *isomorphous provocative reaction*, i.e., the tendency of the patient's organism to react to irritation (mechanical, chemical, etc.) of the skin with an eruption of new psoriatic lesions in that area (Figs. 91, 92, 93). In some cases the progressive stage of psoriasis leads to a disseminated affection of all of the patient's skin—*psoriatic erythroderma*. In these cases the skin becomes saturated red infiltrated, hot to the touch, dry and covered with lamellar whitish or yellowish scales. The patients complain of

chillness, a sense of contraction of the skin, dryness and pain. Psoriatic erythroderma is often accompanied by itching. Some patients exhibit general disorders—fever, disturbed sleep and lack of appetite. Psoriatic erythroderma sometimes involves loss of hair and nails; the joints are affected less frequently.

Lesions in the joints are observed not only in psoriatic erythroderma, but also in cases of usual psoriatic eruptions on the skin (*arthropathic psoriasis*). The affections of the joints vary in severity from comparatively mild rheumatoid pains to phenomena of deforming polyarthritis with contractures and ankyloses.

Treatment. A very important part in the treatment of psoriasis is played, in addition to general therapy, by a proper regimen and elimination of harmful factors from the patient's working and living conditions.

The patient's regimen must provide for at least 7-8 hours of daily sleep, vitamin-rich food and regular outdoor hours (not less than 1.5-2 hours a day). The patient must be very thoroughly examined and, in cases of internal or nervous diseases, must be given corresponding treatment. It is very important to safeguard the patient against any nervous shock.

The *methods of general treatment* of psoriasis depend on the individual characteristics of the patient's organism and the stage of the disease. In the progressive stage and in cases of the stationary stage involving disseminated eruptions and phenomena of dysfunction of the central nervous system (insomnia, itching, loss of appetite, etc.) the following agents are effectively used: intravenously—a 0.25 per cent novocain solution, intramuscularly—1 ml of a 2.5 per cent aminazine (chlorpromazine) solution twice a day, a 10 per cent sodium hyposulfite solution, and thiamine (vitamin B_1), and per os—1 tablespoonful of a 0.25-2 per cent sodium bromide solution 3 times per day and 15-20 drops of vitamin A concentrate 3 times per day after meals for 2-3 months.

Good effects are sometimes produced by autohemotherapy and intravenous administration of a 10 per cent calcium chloride solution.

Good results are also achieved in the progressive stage, especially in psoriatic erythroderma, by blood transfusions—100-200 ml once in 6-7 days (a total of 4-7 transfusions). Corticosteroid hormones are very effective in psoriatic erythroderma and arthropathic psoriasis.

Psoriasis patients also benefit from intramuscular or intravenous injections of 5-10 ml of a 5 per cent ascorbic acid solution. The agents used in general treatment may likewise be employed in the stationary stage, but they often prove less effective.

Arsenicals—Asiatic pills, Fowler's solution, osarsol (acetarsone) and subcutaneous injections of sodium arsenide (adminis-

tered in courses of 1-1.5 months'duration) are also used in the stationary and regressive stages.

In these stages of psoriasis the patients are sometimes helped by peroral administration of a 2-4 per cent potassium iodide solution.

Local treatment of psoriasis is also very important because it favours resorption of psoriatic lesions.

In the progressive stage local, external treatment must be administered cautiously. Strong, irritant ointments may aggravate the process and even cause development of psoriatic erythroderma.

In this stage patients are prescribed nonirritating ointments—skin-greasing ointments, a 2-3 per cent ammoniated mercury ointment, a 2-3 per cent salicylic acid ointment, zinc oil, etc. The patient must take a bath or shower every day or every other day.

Stronger substances favouring resorption of the papules and patches are used for local treatment in the stationary and regressive stages. One of these agents is *psoriasin* (ointment containing nitrogen mustard and vaseline). It is carefully rubbed into the psoriatic lesions once a day for a period of 6 days. On the seventh day the patient is given a bath or shower and a rest, but is not administered the preparation. This cycle of treatment is repeated 5-7 times.

To the patches with considerable infiltration psoriasin may be applied in the form of dressings on sheets of wax-paper.

Psoriasin must not be applied to the face or hairy part of the head because it may get into the eyes and cause severe conjunctivitis.

In some cases psoriasin produces dermatitis and sometimes folliculitides and furuncles. In these cases it must be discontinued. It is best to begin the first cycle (6 days) of psoriasin treatment on a circumscribed focus of affection. As soon as it is ascertained that it produces no irritating effects inunction of the ointment may be started in all psoriatic foci. For relapses of psoriasis this preparation must be prescribed with caution because sometimes patients become hypersensitive to it. In the stationary and regressive stages of psoriasis good effects are produced by ointments containing 5-10 per cent salicylic acid, 10-20 per cent sulfur and 5-20 per cent pitch.

Chrysarobin ointments in gradually increasing concentrations (2-5-10 per cent) are also used. Chrysarobin may cause dermatitis, conjunctivitis and renal irritation, for which reason ointments containing this substance must not be used in the treatment of eruptions on the face and hairy part of the head. Nor may they be applied to all foci at once, but rather to a circumscribed area. Within 5-6 days inunction may be started in another area, etc.

The best physiotherapeutic methods conducive to resorption of psoriatic lesions are sun baths and ultraviolet irradiation by

mercury vapour lamps. Both may be prescribed only to patients with a "winter form" of psoriasis, i.e., those who have remissions during the spring and summer and, contrariwise, relapses and exacerbations of the disease in autumn and winter. Sun baths and mercury vapour lamp irradiation are contraindicated for patients with the "summer form" of psoriasis and a tendency to exacerbation precisely during the spring and summer.

Many psoriasis patients are benefited by sulfur baths. During the warm season the southern health resorts are beneficial only for patients with the "winter form" of the disease. Patients with the "summer form" may go for summer treatment to moderate climate health resorts, and for autumn and winter treatment to southern health resorts.

LICHEN RUBER PLANUS

Lichen ruber planus is a chronic skin disease characterised by a papular eruption without acute inflammatory phenomena and is almost always accompanied by itching. It usually occurs between 20 and 50 years of age, but is also observed in children and old people.

The etiology of lichen ruber planus is not well known.

A. Polotebnov believed the disease to be caused by dysfunction of the nervous system. A. Pospelov and P. Nikolsky also considered disorders of nervous activity to be the cause of lichen ruber planus. Development of the disease following nervous shock, the symmetry of the eruptions and intense itching, the localisation of the eruptions in some cases along the course of the nerves and their concurrence with neuralgias, and the effectiveness of the methods of treatment aimed at eliminating the diseases of the nervous system all indicate the nervous origin of this disease. Some authors, including A. Ukhin and A. Krichevsky, favour the viral etiology of lichen ruber planus.

Lichen ruber planus usually begins with eruptions of papules on the flexor surfaces of the limbs. It is particularly frequently localised on the flexor surfaces of the forearms and wrists, in the area of the elbow bends, axillae and popliteal spaces, on the anterior surface of the shanks, in the lumbar region and on the genitals. In disseminated forms of lichen ruber planus eruptions also appear on any other part of the skin.

The papules of lichen ruber planus have a very characteristic appearance. They are flat and polygonal, and vary in size from that of a pinhead to that of a lentil. Many of them are umbilicated in the centre and are of a purplish-red colour. Under lateral lighting they show a characteristic waxy lustre. The papules do not enlarge much, but owing to their frequent arrangement in groups (Fig. 94) coalesce and form patches even when enlarging a little. The patches of lichen ruber planus may grow to the size of the palm and even larger; they are purplish-violet or greyish-red (Fig. 95). When localised on the anterior surface of the shanks they often exhibit phenomena of hyperkeratosis which lend them a warty appearance (Fig. 96). Through a magnifying glass they appear surrounded by numerous very small papules of the same type, but of the colour of normal skin.

Most patients with lichen ruber planus have rather intense itching, sometimes very stubborn and distressing. On resorption

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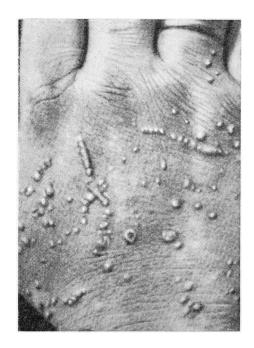
Fig. 94. Lichen ruber planus (from
A. Kartamyshev)
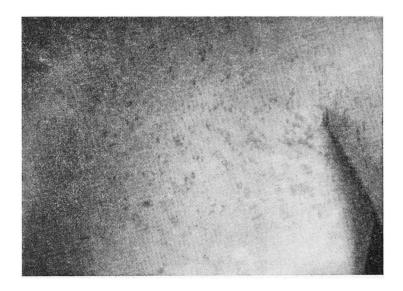
Fig. 95. Disseminated lichen ruber planus

the papules assume a darker, purplish-brown or brownish colouring, and become covered with thin scales. The papules gradually disappear, leaving pigmented macules. The lichen ruber planus papules are stable lesions and never change to other lesions.

Individual papules of lichen ruber planus exist for several weeks, while larger patches may persist for months and even years. In most cases the disease lasts a total of from several months to

Fig. 96. Hypertrophic lichen ruber planus

2-3 years. Remissions and even disappearance of the eruptions are often followed by relapses. In some cases the disease becomes acute, i.e., a large number of papules simultaneously appears on different parts of the skin. The number of papules increases with each passing day and the itching becomes very intense.

Many lichen ruber planus patients show an *isomorphous provocative reaction* to various external irritations of the skin, new papules in the form of bands, arches, etc., appearing at the site of trauma.

The eruptions in lichen ruber planus patients quite often also occur on the oral mucosa. Small whitish papules barely elevated above the level of the surrounding mucosa appear. Patches, bands and rings form as a result of coalescence of the papules. The general appearance of the eruptions on the mucosa resembles a thin, delicate net.

Treatment. One of the most commonly used methods of general treatment of lichen ruber planus patients is intravenous or peroral administration of nicotinic acid. This method is particularly

effective in cases of acute eruptions, secondary erythroderma and very intense itching.

In usual chronic lichen ruber planus favourable effects are in some cases produced by prolonged administration of arsenic (1-1.5 months) subcutaneously or per os.

Lichen ruber planus is known to be successfully treated by influencing the central nervous system. For this purpose novocain is administered intravenously, perorally and in the form of a block, aminazine (chlorpromazine), thiamine (vitamin B_1) intramuscularly or per os, and bromides.

Local treatment consists in application of antipruritic agents in the form of alcohol solutions, pastes and ointments containing pitch (10 per cent), anesthesin (5-10 per cent), menthol (1-2 per cent), carbolic acid (1-2 per cent), dimedrol (3 per cent), and prednisolone (0.5 per cent) or hydrocortisone (1 per cent).

In cases of stubbornly nonresorbing patches of lichen ruber planus paraffin therapy and roentgen therapy are effectively used, the patches are covered with imbricated strips of adhesive plaster or are superficially frozen with carbon dioxide snow. In cases of a prolonged, stubborn course of the disease the patients are greatly benefited by health resort treatment—sulfur baths and pelotherapy.

PITYRIASIS ROSEA

Pityriasis rosea is an acutely developing inflammatory skin disease characterised by scaly reddish-pink macules.

The etiology of pityriasis rosea is not well known. There are reasons to consider it an infectious disease. Some investigators ascribe the role of the causative agent to the streptococcus, but no final proof has as yet been obtained.

This disease most commonly occurs in spring and autumn.

The eruption of the macules is sometimes preceded by indisposition, headache and elevated temperature. In about half the number of cases the disease begins with the appearance of one or less frequently two or three so-called maternal patches in the form of pink-red macules with branny scales and clearly defined borders. The macules are oval or round and vary in size from that of a lentil to that of a large coin and even larger.

The macules gradually increase in size, their central parts assuming a yellow or brownish hue; the skin becomes wrinkled and resembles crumpled cigarette paper. The peripheral parts of the macules remain pink, slightly elevated above the level of the surrounding skin and covered with branny scales. Maternal patches are most commonly localised on the chest, neck, back, and less frequently on other parts of the skin.

Several days after development of the maternal patch there appear numerous pink-red macules, punctate or the size of the little finger-nail, slightly raised above the level of the surrounding skin (Fig. 97).

Subsequently the centre of the macules becomes umbilicated, assumes a yellowish hue, becomes wrinkled and covered with branny scales and the slightly elevated peripheral areola enlarges. Sometimes the centre quickly grows pale and the macules assume an annular form, resembling lockets. The macules enlarge to the size of large coins and sometimes even larger.

The eruption of new macules continues in "spurts" for 2-4 weeks, then ceases and the old macules disappear. In most cases the eruption disappears without leaving a trace 5-6 weeks after the beginning of the disease; sometimes it may last 2 and even 4 months.

Pityriasis rosea is most commonly observed in youths and adults. The disease is often accompanied by itching, in most cases moderate. Only some patients complain of very intense itching.

Treatment. The general treatment of patients with pityriasis rosea consists in administration of sulfonamide preparations for 1-2 weeks, calcium chloride and sodium hyposulfite. Dimedrol, penicillin and autohemotherapy are also prescribed. Indifferent
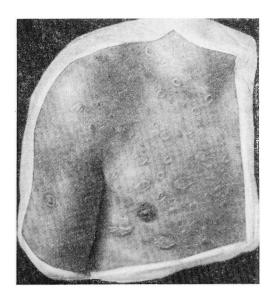
Fig. 97. Pityriasis rosea (from A. Arievich and Z. Stepanishcheva)

suspensions and pastes to which 1-5 per cent ichthyol, 2-3 per cent dimedrol and 1-2 per cent menthol are sometimes added are used for external treatment. Any vigcrous treatment (pitch, sulfur, etc.) is contraindicated because it may easily irritate the patient's skin. Washing with water is also likely to irritate the skin, for which reason it is prohibited during the first 1-2 weeks; after this period the skin may be washed with children's soap, but without a sponge, once in 7-10 days.

DISEASES OF THE CUTANEOUS GLANDS

Disturbances in the functions of the sebaceous and sweat glands occur very often. Diseases of the cutaneous glands are always due to pathologic deviations in the organism.

The most common diseases of the sebaceous glands are seborrhea and acne vulgaris.

Seborrhea

The word *seborrhea* literally means a flow of sebum and indicates an excessive secretion and discharge of sebum by the sebaceous glands.

In cases of hyperfunction of the sebaceous glands the skin is sometimes covered with a layer of sebum and looks oily, glossy, the ostia of the sebaceous glands are visibly dilated and often gape. This disease is called *seborrhea oleosa.*

Seborrhea oleosa is particularly clearly marked in the areas abounding in sebaceous glands—on the nose, cheeks, forehead, hairy part of the head and along the middle line of the chest and back. In these cases the ostia of the sebaceous glands may be plugged with sebum.

On the hairy part of the head seborrhea oleosa is often combined with a loss of hair. The sebum retained and disintegrating on the surface of the skin may cause itching, scratching and irritation of the skin.

In other cases the hyperfunction of the sebaceous glands may be combined with parakeratosis—disturbance in the process of keratinisation of the epidermis.

The secreted sebum impregnates the numerous branny scales and causes a form of seborrhea known as *seborrhea sicca.*

Seborrhea sicca is a very common disease. The pathologic process is most clearly marked on the face and scalp. The skin of such patients appears dry and has many branny scales. The scaling increases by frequent washing and shaving. In many patients affected with seborrhea sicca the skin is easily irritated.

The hairy part of the head is abundantly covered with branny scales which are shed on the patient's clothes when the hair is combed or stroked with the hand. Seborrhea sicca is often accompanied by itching and sometimes pain in the hairy part of the head. When it attacks the scalp it very often causes loss of hair, which begins in the frontal or parietal region and may lead to permanent baldness due to atrophy of the hair papillae (alopecia seborrheica).

In men this process often results in total and permanent baldness of the frontal and parietal regions, and sometimes of the entire head. The skin in the alopecic areas has a smooth, shiny surface, the layer of subcutaneous adipose tissue becomes thin and the mobility of the skin on the bony base is limited. These changes show that seborrhea is a disease conditioned by considerable changes in the organism.

Seborrhea usually begins at the time of sexual maturation. Acute infectious diseases and diseases of the nervous system, the internal organs and endocrine glands, chronic intoxications, nervous shock and overstrain unfavourably affect the course of seborrhea and expedite alopecia seborrheica. The causes and mechanisms of seborrhea are still unclear.

Hypofunction of the sebaceous glands (asteatosis) is observed in old age, after severe diseases, in malnutrition, certain skin diseases (psoriasis, ichthyosis) and under the influence of noxious occupational factors which cause defatting of the skin (work with alkalis, organic solvents, constant humidity and maceration of the skin). The skin becomes dry, inelastic and fissured, especially on the dorsal surface of the hands and the extensor surface of the joints.

Treatment. The treatment of seborrhea may be effective only if the unfavourable changes in the organism conditioning the disease in the given patient are discovered and eliminated. Sometimes the treatment of seborrhea is greatly aided by treatment of the concurrent gastrointestinal disease. In other cases the treatment has to be confined to measures aimed at diminishing the phenomena of seborrhea and impeding its development. In many cases this treatment administered systematically produces good results.

The following agents are prescribed for the general treatment of seborrhea patients: riboflavin (vitamin B_2), vitamin B_6 and vitamin A; washed sulfur per os; endocrine preparations.

The best agent for external treatment of seborrhea is selenium sulfide or sulsen (selsun). It is dispensed as a paste, suspension (lotion), cream and soap usually containing 2.5 per cent sulsen.

For the treatment of seborrhea sicca sulsen paste or a sulsen suspension (lotion) is used. The patient must first wash his head with warm water and overfat soap (children's lanolin, spermaceti). Following this about 1 teaspoonful of sulsen paste is rubbed into the skin of the hairy part of the head until a foam is formed. About 5-10 minutes later the paste is washed off with warm water (without soap) and the head is dried with a towel. The sulsen paste is rubbed in once a week over a period of 4-6 weeks. The sulsen suspension (lotion) and cream are used in the same manner.

Mainly sulsen soap is used in the treatment of seborrhea oleosa. The patient first washes his head with warm water and alkaline

soap (for example, 72 per cent laundry soap) and then, without drying his hair, lathers his head with sulsen soap, thoroughly rubbing it into the skin of the hairy part of the head, which requires about 2-3 g of soap. The sulsen soap is then washed off with warm water and the head is dried with a towel. Sulsen soap is also used once a week, although during the first two weeks it may be used twice a week. The treatment takes 4-6 weeks.

Sulsen preparations must not be allowed to get into the eyes; after handling these preparations the hands must be washed.

After the course of treatment the use of sulsen preparations is recommended once in 1-2 months for prophylactic purposes. In the event of a relapse the course of treatment may be repeated.

Applications of alcohol solutions containing sulfur (3-6 per cent), resorcinol (1-2 per cent) and pitch (1-3 per cent) are used for local treatment of seborrhea oleosa.

Suspensions and ointments containing sulfur (1-5 per cent), pitch (1-2 per cent), castor oil (5-20 per cent) and other preparations are used for local treatment of seborrhea sicca.

Acne

Common blackheads or acne vulgaris is a widespread disease. Blackheads most frequently appear in youths affected with seborrhea. The process begins with formation of sebaceous plugs or comedones in the ostia of the sebaceous glands. The obstruction of the sebaceous glands leads to retention of their secretion in the glands, distention of their cavities and disintegration of the secretion. Staphylococcal infection very frequently accompanies this condition and causes an inflammatory process in the sebaceous glands and the adjacent tissues.

Acne vulgaris is localised on the face, in the region of the sternum and on the back (on the scapulae and between them). Red or purplish-red follicular papules form in these areas. The papules vary in size from that of a millet seed to that of a pea. In the centre of each papule there is a sebaceous plug. The papules exist from a few days to 2-3 weeks and then disappear leaving a temporary pigmentation. But owing to the suppuration caused by the staphylococcus some of the papules develop into pustules. The pustules are of conical or semispherical shape, are surrounded by an infiltrated red or purplish-red areola and on healing often leave fine scars. In some patients acne involves formation of nodes extending to the subcutaneous cellular tissue. In cases of concurrent staphylococcal infection these nodes may develop into abscesses. Upon healing such abscesses leave larger and deeper scars.

Acne vulgaris is a chronic disease because new papules and pustules continuously appear. The process may last several years. The blackheads appearing at the time of sexual maturation disap-

pear in most patients at 20-25 years of age. In some patients, however, they may persist for a longer time. Chronic acne is particularly frequently observed in patients with a tendency to formation of deep infiltrated nodes and large cysts of the sebaceous glands which often suppurate.

Treatment. For effective treatment of acne vulgaris it is important to eliminate the extant pathologic deviations in the organism and to establish an appropriate regimen for the patient, namely, adequate sleep, regular meals, fresh air and sports. It is necessary to treat the foci of chronic infection in the teeth, gums, tonsils, etc.

In many patients the course of the disease is favourably affected by regulation of gastrointestinal function. Preparations of sex hormones (methyltestosterone, sinestrol [diethylstilbestrol dipropionate], folliculin, etc.), autohemotherapy, injections of laky blood, milk, vitamins C, A, B_1, B_2 and B_6 and washed sulfur per os are successfully administered in the treatment of acne. Local treatment of the disease consists in applications of scaling and anti-inflammatory agents—salicylic acid (3-10 per cent), resorcinol (3-5 per cent), sulfur (2-10 per cent), camphor (5-10 per cent) and ichthyol. These agents are used in the form of ointments or alcohol solutions. Favourable effects are produced by irradiation of the affected areas with erythema doses of ultraviolet rays (mercury vapour lamp).

In cases where patients have many pustules indicating active staphylococcus infection good results are often produced by peroral administration of tetracycline, terramycin and biomycin (chlortetracycline). As for external treatment, such patients often benefit by rubdowns with an alcohol synthomycin solution and applications of ointments containing synthomycin and biomycin.

In cases of deep infiltrated nodes good effects are produced by paraffin therapy.

DISEASES OF THE SWEAT GLANDS

Disturbances in the function of the sweat glands are most commonly manifested in hyperidrosis or hypohidrosis, the former occurring much more frequently than the latter. Disturbances in perspiration are often the result of dysfunction of the central nervous system. It is well known that under emotional stress, fright or pain sweat appears on the face, the palms and other parts of the skin.

Hyperidrosis may be *general* and *local*. General hyperidrosis is observed in febrile diseases, hard emotional experiences and certain diseases without elevated temperature (pulmonary tuberculosis, etc).

In local hyperidrosis excessive sweating is most commonly observed on the palms and the soles of the feet, as well as in the axillae and the perineum. The skin on the palms and soles of the feet becomes moist, sticky, hot and sometimes cold to the touch. Marked hyperidrosis of the palms may greatly inconvenience the patient and make it impossible for him to do certain kinds of work (mechanical drawing, etc.).

If the patient does not wash regularly, hyperidrosis of the feet is accompanied by an unpleasant odour (brcmidrosis).

Hyperidrosis of the feet predisposes to epidermophytosis and is one of the main causes of *excoriation* and chafing of the feet; continuously moist and often macerated (loosened) by sweat the skin is more easily injured mechanically and irritated by the disintegrating sweat.

Hyperidrosis of the axillae is often complicated by hidradenitis and eczematisation. Hyperidrosis of the perineum often gives rise to chafing in the intergluteal fold.

Treatment. Elimination of unfavourable factors from the patient's everyday life, adequate rest, fresh air, morning setting-up excercises and sports create favourable conditions for effective treatment of hyperidrosis.

Novocain and vitamin B_1 are used for general treatment.

Dusting with urotropin powder is prescribed for local treatment of hyperidrosis of the feet (see p. 327). In hyperidrosis of the palms the skin of the palms is painted with a 3-5 per cent formalin solution in 140 proof alcohol.

Patients with hyperidrosis of the axillae are advised to cut short or shave the hair in this region, to wash the axillae with soap and water and dust them with boric acid powder or the fol-

lowing mixture—1.0 of salicylic acid, 9.0 of powdered boric acid and 5.0 of talc.

It is very important to *prevent excoriation of the feet*, because this condition affects the patient's working capacity and may temporarily even incapacitate the patient.

In addition to hidrosis of the feet, excoriation is also caused by improperly fitting footwear (footwear not corresponding to the size and shape of the feet), defects of footwear (protruding nails, etc.), improper and defective socks and stockings (torn, having heavy seams, etc.).

Prevention of excoriations of the feet consists in:

(1) timely repair of footwear and change of socks and stockings;

(2) wearing proper socks and stockings;

(3) keeping the feet strictly clean—washing them with soap and water every night before retiring;

(4) treatment of the hidrosis of the feet.

MALIGNANT TUMOURS OF THE SKIN

Malignant tumours of the skin may be derived from the epithelium, pigments or connective tissue. The most common epithelial tumours are called *epitheliomas* or cancer of the skin.

Cancer of the skin most frequently affects middle-aged and old people. It is also well known that cancer of the skin often develops in areas formerly acted upon by various external irritants—sunlight, roentgen rays, radioactive radiations, pitch and certain chemical substances.

In most patients development of epithelioma of the skin and mucous membranes is preceded by chronic inflammatory and dystrophic processes in these areas. When these inflammatory and dystrophic changes exist for a long time they assume the character of *precancerous states* which are characterised by intensified multiplication of epithelial cells, increased number of cells with dividing nuclei, disturbed keratinisation and appearance of atypical cells.

Precancerous diseases are a transition to the development of epithelioma. The transition is usually gradual. Precancerous states may vary in their clinical picture. Often they have the appearance of a focus of disturbed keratinisation, especially in elderly people, i. e., they represent circumscribed yellowish or brownish macules or papules with a thickened and loosened stratum corneum. Scraping reveals scales firmly adhering to their base. Attempts at removing the scales easily cause slight bleeding. In other cases precancerous states constitute a chronic inflammatory process on the vermilion border (cheilitis), unhealing ulcer at the site of a survived disease (pyoderma, etc.) or trauma. On the oral mucosa precancerous diseases may appear as whitish macules— foci of degeneration and keratinisation of the stratified epithelium of the mucosa (leukoplakias and leukokeratoses).

Development of precancerous diseases and their transition to cancer are favoured by atrophic changes in the skin and mucosa in old age, excessive solar irradiation, constant mechanical irritations (for example, irritation of the oral mucosa by the sharp edge of a tooth), smoking and chemical irritations (pitch, products of coal distillation, etc.).

Two principal forms of epithelioma of the skin are distinguished: basal-cell epithelioma composed of cells of the basal layer of the epidermis, and prickle-cell epithelioma composed of cells of the

Fig. 98. Basal-cell epithelioma (from
A. Kartamyshev)

Fig. 99. Prickle-celi epithelioma

prickle-cell layer of the epidermis or similar cells of the epithelium of the mucous membranes.

Basal-cell epithelioma. The disease begins with formation of a yellowish-waxy or pearl-grey nodule varying in size from that of a pinhead to that of a pea. It is most commonly localised on the face, especially, the nose, eyelids, temples and forehead. The nodule slowly enlarges, sometimes showing no appreciable changes for years. Often several nodules appear and gradually coalesce. In time a dense patch is formed with a sanguineous crust in the centre; the crust is usually repeatedly torn off by the patient, but each time reforms, becoming larger and thicker. After a while, usually several months and sometimes years, the patch becomes ulcerated. The sizes and depths of the ulcers vary and for the most part gradually increase (Fig. 98). The ulcer floors are uneven, readily bleed and are often covered with sanguineous crusts. A pearl-grey elevation which sometimes looks like a strand is well seen along the edge of the ulcers.

Basal-cell epithelioma is a relatively benign form of cancer of the skin. It runs a prolonged, slow course; no metastases are, as a rule, observed. However, existing for a long time and untreated basal-cell epithelioma may undergo deep ulceration and assume a malignant course with lethal results.

Prickle-cell epithelioma. This form of cancer of the skin and mucous membranes is marked by a much faster development and malignant course. It begins with the appearance of warty outgrowths or papules on the skin and papillae on the mucous membranes. These lesions very rapidly enlarge and coalesce with the result that a patch or node forms and soon, usually a few months after the onset of the disease, becomes ulcerated. The ulcer has dense, everted edges and an uneven, bleeding floor often covered with growths (Fig. 99). In many cases foci of keratinisation (epithelial pearls) and layers of keratic tissue (keratinising cancer) are seen on the ulcer floor.

Prickle-cell epithelioma is localised on the oral mucosa, near natural orifices, on the face, lower lip, genitals and limbs. The ulcer comparatively rapidly grows larger and deeper and inflicts great destruction. It often causes intense pain. Metastases of the cancer to the nearest (regional) lymph nodes are frequently observed; the lymph nodes become dense, coalesce and may ulcerate.

In the absence of proper treatment prickle-cell epithelioma leads to cancerous cachexia and death.

Treatment. Success in the treatment of cancer of the skin depends on early diagnosis. Timely diagnosis favours good therapeutic results. In cases of late diagnosis the treatment of cancer of the skin offers great difficulties. In neglected cases of prickle-cell epithelioma the treatment often fails to save the patient from cachexia and death.

In precancerous states it is necessary to treat the inflammatory processes, chronic ulcers and disturbances in keratinisation. If these therapeutic measures prove ineffective over a period of several weeks, the entire precancerous focus is removed surgically. The best agents in the treatment of basal-cell epithelioma are surgical excision, roentgen rays, radium and radioactive isotopes. In prickle-cell epithelioma radiation therapy is much less effective, and excision of the tumour is the principal method of treatment. Radiation therapy is often prescribed after the operation.

Melanoma (melanocarcinoma, melanosarcoma) occurs less frequently than epithelioma of the skin. Melanoma is a tumour composed of pigmented cells. It is supposed that the pigmented cells are modified cells of nerve sheaths. Melanoma usually develops from late pigmented macules (birthmarks) appearing on the skin after sexual maturation and sometimes even from pigmented macules, either inborn or appearing during the first years of life. Development of melanoma from inborn or early pigmented macules is for the most part due to their irritation—cauterisation, cutting-off, tearing-off, picking, etc.

Transformation of a pigmented macule into a melanoma is evidenced by the changes in its appearance—it begins to enlarge and rises above the level of the surrounding skin. Scales and crusts appear on its surface, and it readily bleeds. Subsequently an ulcer with eroded edges and an uneven floor is formed. Melanoma is characterised by rapid development. Metastases into regional lymph nodes and internal organs soon appear, cachexia develops and death ensues. Melanoma is the most malignant tumour of the skin and one of the most malignant tumours of the human organism.

Treatment of melanoma consists in early excision of the tumour with an extensive portion of adjacent healthy skin and regional lymph nodes. Radiation therapy is used mainly in the form of short-focus roentgen rays.

Prevention of malignant tumours of the skin and mucous membranes is based on prophylactic examinations of the population, extensive health education, early diagnosis and proper treatment of precancerous diseases of the skin and mucous membranes.

VENEREAL DISEASES

SYPHILIS

Infectious diseases acquired through sexual intercourse are called venereal diseases. The term *venereal* is derived from Venus, the ancient Romans' goddess of love.

Under certain conditions venereal diseases may also be acquired nonvenereally, i.e., outside of sexual intercourse. The most common venereal diseases are syphilis, gonorrhea and chancroid.

* * *

Syphilis is a chronic infectious disease caused by the *Treponema pallidum* and capable of affecting all organs and systems of the human organism. The disease is transmitted through sexual intercourse, as well as through other forms of direct and indirect contact of healthy people with patients. It may also be transmitted from mother to child during the intrauterine period.

Syphilis has been known a long time. Ancient Indian, Chinese, Hebrew and Greek manuscripts which have come down to our time contain descriptions of pathologic phenomena resembling syphilis. However, very little was known about this disease, it was frequently confused with other diseases and many ancient descriptions are vague and contestable. At the end of the 15th century, very soon after the discovery of America by Columbus, an epidemic of syphilis with a severe course developed in Europe. The connection between the extensive spread of syphilis and the return of Columbus' expedition from America gave many scientists the idea that the disease was first brought to Europe from America by Columbus' seamen who had contracted it there from Indian women. But the "American" origin of syphilis is at variance with the results of excavations of ancient burial grounds. The bones of some people who had died and were buried long before Christ bear signs of gummas.

The question of whether syphilis is of ancient or American origin has not yet been finally settled.

The *causative agent of syphilis*, the *Treponema pallidum*, is a spiral organism. It is 6-20 μ long ($1\mu=0.001$ mm) and $1/4$-$1/3$ μ thick: the number of its spirals varies from 8 to 12 and even 20 (Fig. 100).

Because it is barely stainable the causative agent of syphilis has been given the designation of *pallidum* (pale). To examine the *Treponema*, dark-field illumination is used. In the dark field the *Treponema* appears as a light spiral possessing complex
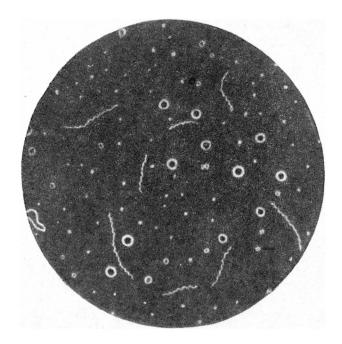
Fig. 100. *Treponema pallidum* in dark-field illumination
(from V. Predtechensky)
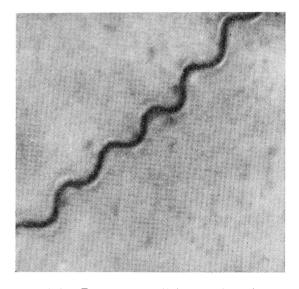
Fig. 101. *Treponema pallidum* under electron
microscope (from multivolume manual on dermatology
and venereology)

motility—translational (back and forth), rotatory (about the axis), flexural and undulating. The complex character of movements makes it possible to distinguish the *Treponema pallidum* under the microscope from other, similar *Treponemaceae* (Fig. 101).

Outside the human organism the *Treponema pallidum* is non-resistant to unfavourable external influences. It is easily destroyed by desiccation and direct sunlight. Heated to 40°C outside the human organism it loses its pathogenicity. At 48°C the *Treponema pallidum* is destroyed in 30 minutes, at 60°C—in 3-5 minutes, and at 100°C—instantly. It tolerates low temperature better— at 10°C it takes 3 hours to destroy it. In the tissues of corpses kept in a refrigerator the *Treponema* retains its pathogenicity for 48 hours. Workers of morgues often acquire syphilis through autopsies. Outside the human organism disinfectants rapidly destroy the *Treponema*. Mercury bichloride kills the *Treponema* instantly in a 1:3,000 concentration. The surface of the skin may be disinfected by washing with soap and water.

In banked blood the *Treponema* can live up to 4-5 days and remain pathogenic (N. Vedrov). In cases of inadequate examination of donors and check-up on their health syphilis may be transmitted through transfusion of the blood of a syphilitic donor.

Normally syphilis occurs only in man and is not observed in any animals. In 1903 the great Russian scientist I. Mechnikov and the French scientist P. Roux succeeded in inoculating the chimpanzee with syphilis. Other monkeys were successfully infected with syphilis by the Russian scientist D. Zabolotny and the German scientist Neisser. Later other scientists managed to inoculate rabbits and white mice with syphilis. Experimental infection of animals has played an important part in the studies of the conditions under which syphilitic infection takes place and develops, as well as in the elaboration of new methods of treating syphilis.

Routes and methods of infection. A patient with active infectious manifestations of syphilis is a source of syphilitic infection. The discharge of the papules, erosions and ulcers of these patients contains a large number of *Treponema pallidum*.

The main route of infection is sexual intercourse. The sexual route accounts for 95-98 per cent of transmission of syphilis.

The nonvenereal route of infection accounts for cases of sanitary ignorance and failure to observe rules of hygiene. In both venereal and nonvenereal transmission of syphilis the disease may be called *acquired* to be distinguished from *congenital* which is transmitted to the child in utero.

Nonvenereal infection may take place directly and indirectly. Direct infection occurs through close contact of a syphilitic patient with a healthy person. Transmission of the disease through kissing, bites, the harmful habit of licking a foreign body out of

the eye and feeding the breast to somebody else's child may serve as examples of direct nonvenereal infection with syphilis.

Indirect or intermediate infection with syphilis is transmission of the disease through things used by a syphilitic patient and contaminated with his discharges. An important negative part in spreading the disease nonvenereally may be played by the habit of eating from common utensils, using common spoons and common drinking cups, and finishing other people's cigarettes or pipes. Children taking fifes and whistles from each other's mouths and playing with common toys may also contract the disease from each other.

Under certain conditions indirect transmission of syphilis may be *occupational.*

For example, shoemakers who had the bad habit of taking shoe-nails into the mouth while working spread the disease in this manner since they put the unused nails back into a common box from which they could be taken by other shoemakers.

There have been cases where medical workers failing to observe the rules of instrument disinfection and sterilisation infected their patients by using contaminated instruments.

The *Treponema pallidum* nearly always gains entrance into the organism through the skin or mucous membranes. Only in special cases may it directly enter the blood stream (infection through blood transfusion from a syphilitic donor).

Most scientists hold that the *Treponema pallidum* may gain entrance into the depth of the skin and spread through the organism only in cases of disrupted continuity of the skin.

Undamaged skin is an insurmountable barrier for the *Treponema pallidum*. But as slight and imperceptible to the eye the injury to the skin may be it is enough to admit the *Treponema pallidum.*

Penetration of the *Treponema pallidum* through the mucous membranes is a different matter. This organism is apparently also capable of penetrating through intact mucosa.

General course of syphilis. Syphilis becomes a general disease at the very moment of infection. It has been shown by experiments on animals that already 30 minutes after inoculation of the rabbit with syphilis the *Treponema pallidum* may be found in the adjacent lymph nodes. Experiments on monkeys have shown that even excision of a large portion of tissue at the point of inoculation and around it 8 hours after inoculation of such an animal with syphilis fails to prevent the animal from developing the disease. During the very first hours and days after inoculation the *Treponema pallidum* multiplies and moves through the intercellular and lymph spaces to the lymph nodes and blood.

But the first signs of syphilis visible to the patient and those around him appear much later—3-3.5 weeks after infection with

the disease. The time elapsing between the moment of infection and appearance of the first signs of the disease is called the incubation period.

A small, dense and painless erosion or ulcer—*primary lesion, ulcerating sclerosis* or *hard chancre*—appears at the point of infection at the end of the incubation period. The primary stage of syphilis (syphilis I) begins at this moment and lasts 5-7 weeks. It would be more fitting, however, to regard the primary stage of syphilis as beginning at the moment of inoculation with the disease.

The primary stage of syphilis is the period of mass multiplication of the *Treponema pallidum*, its spread through the organism and gradual reorganisation of the organism's reactivity. Formation of the primary lesion is the first sign of the changes beginning in the organism's reactivity.

Multiplication of the *Treponema pallidum* at the site of its entrance and its movement through the nearest lymph spaces begin in the first hours and days after inoculation, whereas the primary lesion appears only within 3-3.5 weeks. This is due to the fact that in the struggle of the human organism against the *Tr ponema pallidum* the properties of the organism, its ability to resist the spread and multiplication of the *Treponema pallidum* are altered. On the other hand, the properties of the *Treponema pallidum* also alter; it adapts itself to the new conditions of existence and begins to multiply intensively.

The signs of altered reactivity during the primary stage of syphilis are an enlargement of the nearest lymph nodes, appearance of a positive Wassermann and flocculation tests, and, lastly, enlargement of all lymph nodes.

The total duration of the primary stage of syphilis, including the incubation period and the time elapsing between the appearance of the primary lesion and the beginning of the secondary stage is usually 8-10 weeks.

Towards the end of the primary stage of syphilis the *Treponema pallidum* penetrates into the blood ever more often and in increasingly greater numbers; the blood carries it to all organs and tissues.

The increasing changes in the reactivity of the organism manifest themselves in a new form—multiple eruptions on the skin and mucous membranes. The appearance of these eruptions starts the *secondary stage* of syphilis (recent syphilis II). The eruptions of the secondary stage are characterised by a comparatively superficial infiltrate and run a benign course, soon disappearing without leaving a trace even in the absence of treatment. Another important characteristic of these eruptions is a large number of the *Treponema pallidum* in the tissue of the eruptive lesions. Like the primary lesion, the eruptions of the secondary stage are highly contagious.

The disappearance of the first eruptions of the secondary stage is followed by a rather long interval with no active manifestations of the disease. This is due to the increase in immunity and destruction of some of the *Treponema pallidum*. However, in the absence of treatment not all the microbes are destroyed and some of them persist in the lymph nodes, at the site of the primary lesion, in various organs and tissues, adapting themselves to the altered conditions of existence in the organism. During the latent stage of the disease (latent syphilis II) the *Treponema pallidum* becomes less active in these foci. But various unfavourable changes in the organism, dysfunction of the nervous system, concurrent diseases and chronic intoxications may cause a decrease in the organism's immunity. Then the microbes preserved in their "depots" become active again, begin to multiply intensively and to penetrate into the blood. New eruptions on the skin and mucous membranes—so-called relapse of syphilis (relapsing syphilis II)—appear as a result. There may be several such relapses. The total duration of the secondary stage is in most cases 2-4 years, more rarely—7-10 years.

If the patient with secondary syphilis is given proper anti-syphilitic treatment, he does not, as a rule, have relapses of the disease.

In the absence of treatment some syphilitics develop within 3 years or somewhat later symptoms of *tertiary syphilis*. This stage of the disease is characterised by formation of tubercles and gummas on the skin and the mucous membranes (active syphilis III). The tubercles and gummas considerably differ in structure and results from the eruptions of the secondary stage of syphilis. They are characterised by development of a deep infiltrate with a *malignant* course, i.e., a tendency to disintegration and replacement by scar tissue. The tertiary stage is also characterised by a small number of the *Treponema pallidum* in the infiltrate of the tubercles and gummas for which reason this stage is comparatively less contagious. The tertiary lesions of syphilis are usually few. During this stage it is the bones and internal organs that are often affected. All these characteristics of the tertiary stage of syphilis denote new changes in the reactivity of the patient's organism. During this stage the disease takes an unfavourable course.

The tertiary stage of syphilis may last several years, sometimes several decades. Active tertiary symptoms may alternate with intervals of latency (latent syphilis III). A small number (up to 5 per cent) of syphilitic patients may, in the absence of treatment or in cases of inadequate treatment, develop 8-15-20 years after inoculation, late diseases of the central nervous system—cerebral lesions or *general paresis* and spinal lesions or *tabes dorsalis*.

These forms of neurosyphilis are characterised by a sharp diminution in the reactivity of the organism.

The affected tissue of the nervous system (the cerebral substance in general paresis and the spinal cord in tabes dorsalis) contains a rather large number of the *Treponema pallidum*; the inflammatory reaction is very weak and phenomena of degeneration and necrosis prevail.

According to J. Jadassohn, German dermatologist (1929), 3 per cent of all syphilitic patients develop tabes dorsalis, 5 per cent— general paresis, and 3 per cent—late meningovascular syphilis.

The U.S.S.R. has a single classification of syphilis.

CLASSIFICATION OF SYPHILIS

1. Seronegative syphilis I—lues I seronegativa.
2. Seropositive syphilis I—lues I seropositiva.
3. Latent syphilis I—lues I latens.
4. Recent syphilis II—lues II recens.
5. Relapsing syphilis II—lues II recidiva.
6. Latent syphilis II—lues II latens.
7. Active syphilis III—lues III activa.
8. Latent syphilis III— lues III latens.
9. Latent syphilis—lues latens.
10. Congenital early syphilis—lues congenita praecox.
11. Congenital late syphilis—lues congenita tarda.
12. Congenital, latent syphilis—lues congenita latens.
13. Early neurosyphilis (duration of the syphilitic infection of up to 5 years).
14. Tabes dorsalis.
15. General paresis paralysis progressiva.
16. Visceral syphilis lues visceralis.

PRIMARY SYPHILIS

In most patients the incubation period of syphilis is 3-3.5 weeks. In much fewer cases it may be shorter (9-10 days) or longer (2 months and longer). The duration of the incubation period depends on the general condition of the organism and the individual reactivity of the patient. It may be prolonged to 3-6 months if the patient was treated with penicillin or another antibiotic for some disease (gonorrhea, angina, etc.) after inoculation with syphilis.

At the end of the incubation period a *primary lesion* or *hard chancre* forms at the site of the inoculation.

Genital and *extragenital* chancres are distinguished, according to localisation. Genital chancres account for about 90 per cent of all cases of primary syphilis. (P. Grigoriev, 1938). However, a large number of extragenital chancres is actually associated with venereal inoculation. Such are the hard chancres in the region of the pubes, abdomen and thighs. These chancres are also called *paragenital* chancres.

Genital chancres most commonly occur on the glans penis and prepuce in men and on the labia and perineum in women.
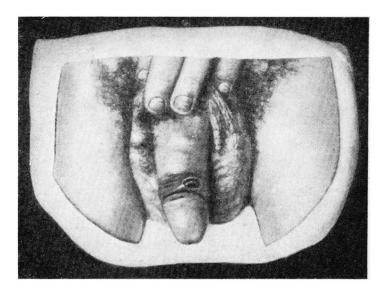
Fig. 102. Hard chancre of the penis (from the Museum of the
Central Institute of Skin and Venereal Diseases)
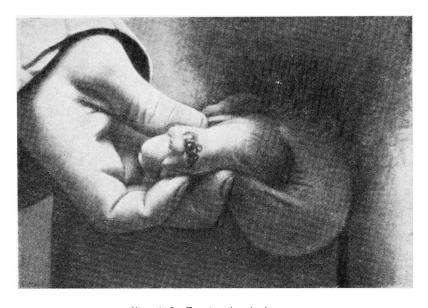
Fig. 103. Erosive hard chancre

Fig. 104. Erosive hard chancre
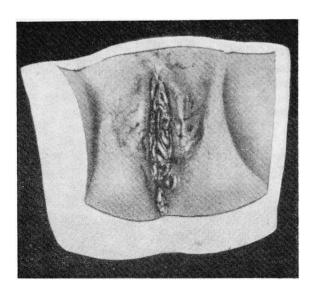
Fig. 105. Hard chancres of the female external
genitalia (from the Museum of the Central Institute
of Skin and Venereal Diseases)
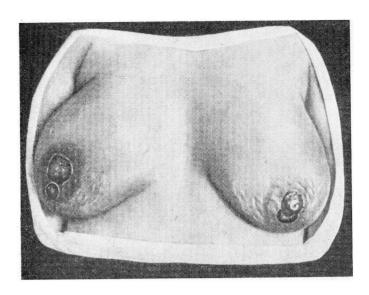
Fig. 106. Hard chancres of the breasts (from the Museum of the
Central Institute of Skin and Venereal Diseases)
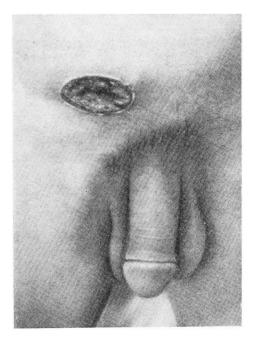
Fig. 107. Giant hard chancre (from A. Zenin and
N. Torsuyev)

Extragenital chancres may occur on any part of the skin and mucous membranes. Hard chancres are particularly frequently observed on the lips and tonsils. In connection with the rise in the cultural and health education standards of the population extragenital hard chancres are a very rare occurrence today.

The primary lesion or hard chancre is an erosion, less frequently a superficial round or oval ulcer varying in size, most commonly from that of a lentil to that of a 10-cent coin. However, there are also very small and very large, giant chancres, sometimes reaching the size of a large coin (see Fig. 107).

In primary lesions there are no acute inflammatory phenomena, the skin or mucosa surrounding the chancre retaining its normal appearance. The edges of the primary lesion are somewhat higher than the floor and descend to the floor gradually, gently, which lends to the erosion or ulcer of the hard chancre a characteristic saucerlike appearance (Figs. 102-107 and 111).

In typical cases the colour of the primary lesion is either meat-red or greyish-yellowish ("colour of spoiled fat"). Because of the scant serous discharge the hard chancre appears shiny, as though lacquered. A dark-field examination of a drop of this discharge reveals the *Treponema pallidum*. The primary lesion often seems raised above the level of the surrounding skin because of the plentiful infiltrate in the base of the lesion elevating it above the level of the surrounding skin. A clearly defined infiltrate is felt when the hard chancre is grasped on two sides with the fingers. The dense elastic consistency of this infiltrate, often resembling the consistency of a cartilage, is very typical. This induration of the floor of the erosion or ulcer served as the reason for the term "hard chancre" or "primary lesion".

As a rule, the primary lesion is not accompanied by subjective sensations. The superficial character and small size of the hard chancre, as well as the absence of pain, are often responsible for the fact that the patient fails for some time to notice the onset of the disease or dismisses it as a "trifle".

The hard chancre most commonly appears singly (in 70 per cent of the cases); less frequently there are two chancres and still less frequently—three. Very many hard chancres very rarely occur in one patient.

Multiple chancres are the result of simultaneous penetration of the *Treponema pallidum* in several parts of the skin or the mucosa and are most commonly observed in debilitated people.

Atypical hard chancres. The clinical picture of a primary lesion in atypical cases is considerably altered. Sometimes, usually when the lesion is localised on the lips, the male prepuce and the labia majora, the primary lesion assumes the form of an *indurative swelling*. The affected area greatly enlarges and becomes purplish-brown and is of a densely elastic consistency.

When localised on the fingers the primary lesion sometimes exhibits considerable peculiarities. In such cases it is called a paronychial chancre because, unlike usual chancres, it is intensely painful and is accompanied by edema and infiltrate of the entire terminal phalanx, thereby simulating usual paronychias. The ulcer forming in such cases often scarcely resembles the hard chancre, has uneven edges and irregular form and is covered with a purulent discharge. The diagnosis of syphilis facilitates discovery of the concurrent bubo—greatly enlarged, densely-elastic ulnar lymph node.

On the tonsils the primary lesion may appear in the form of an erosion, ulcer and amygdalitis. In these cases the diagnosis is rendered somewhat difficult by the resemblance of the affection to the clinical picture of angina; the resemblance is particularly great in cases of chancre-amygdalitides. The affected tonsil is greatly enlarged, red, edematous and painful, and protrudes into the lumen of the fauces. Swallowing and sometimes speech are rendered difficult; elevated temperature and headache are often observed. A correct diagnosis of syphilis is usually aided by the affection of a single tonsil, whereas in ordinary anginas both tonsils are mostly affected. The presence of densely-elastic cervical and submaxillary lymph nodes also facilitates the diagnosis of syphilis.

In the discharge of the primary lesion on the tonsil taken by means of a platinum loop it is possible to discover the *Treponema pallidum* and thereby to establish a timely diagnosis.

Hard chancre may become complicated by secondary pyogenic infection, in which cases the surrounding skin becomes bright-red and edematous and the floor of the chancre is covered with a copious purulent discharge.

In men's genital chancres the secondary infection may take the form of *balanoposthitis* in which the glans penis and the inner fold of the prepuce become hyperemic, edematous and covered with numerous superficial irregularly-shaped erosions with a purulent discharge. Women may develop *vulvitis* with similar phenomena in the region of the labia majora and minora. The concurrence of secondary infection usually also produces subjective sensations, namely, burning, tension, sometimes pain and itching.

In men with a long and narrow prepuce hard chancre is often complicated by *phimosis* or *paraphimosis*. In phimosis it is impossible to retract the prepuce and uncover the glans penis (Fig. 108). In paraphimosis the glans penis is constricted by the retracted prepuce, which gives rise to edema, pain and sometimes superficial necrosisof the tissues (Fig. 109).

Still more serious complications of the primary lesion are *gangrenous* and *phagedenic* chancres. Gangrenous chancre is characterised by formation of a dark-brown or black membrane consisting

Fig. 108. Phimosis (from M. Zheltakov)
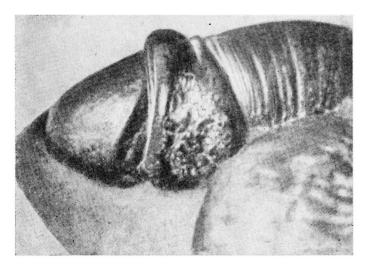
Fig. 109. Paraphimosis (from M. Zheltakov)
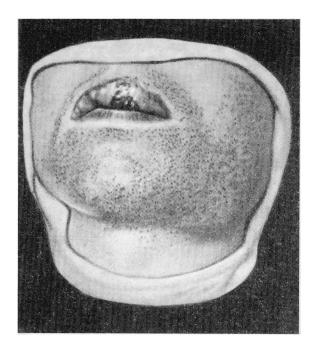
of necrotic tissue on the floor of the lesion. Gangrenous chancre heals slowly and always leaves a scar. In phagedenic chancre the necrosis of the tissues is not confined to the floor of the primary lesion, but rapidly spreads in depth and to the adjacent parts of the skin. Most of the time the patient runs a fever. Gangrenous and phagedenic chancres are most commonly the result of concurrent anaerobic infection. Phagedenic chancre is usually observed in debilitated and emaciated persons, in old people and alcoholics.

Complications of the primary lesion render early diagnosis of syphilis extremely difficult not only because they make the clinical picture of the affection untypical, but also because in the presence of a secondary infections it is very difficult to discover the *Treponema pallidum* in the discharge from the lesion.

In most cases the primary lesion exists till the secondary stage of syphilis. Large lesions with a great deal of infiltrate not infrequently persist for weeks even after the eruptions of the secondary stage, whereas lesions with a leaflike infiltrate often heal soon after their appearance.
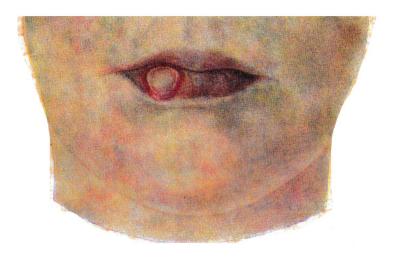
Fig. 111. Hard chancre of the lower lip (from multivolume manual of dermatology and venereology)

If the primary lesion is an erosion, it heals without leaving a trace. Healing of an ulcerated lesion leaves a scar.

Syphilitic scleradenitis or concurrent bubo. From 7 to 10 days after the appearance of the primary lesion the adjacent lymph nodes become enlarged. If the lesion is localised in the region of the genitals the inguinal nodes enlarge on one and in a few days also on the other side. In extragenital chancres those lymph nodes enlarge which collect the lymph from the given part of the skin (Fig. 110).

The lymph nodes enlarge to the size of a bean and sometimes larger, have an elongated form, a densely-elastic consistency and smooth surface, and do not adhere to each other, to the surrounding tissues or the skin covering them. The skin over the lymph nodes retains its normal colour. There are no phenomena of acute inflammation, the lymph nodes hardly ever suppurate and cause no pain.

In cases of complication of the primary lesion by secondary infection the lymph nodes adhere to each other, to the surrounding tissues and the skin, and become painful.

From 2 to 3 weeks after the appearance of the primary lesion the serology tests (Wassermann test, flocculation test) change from negative to positive. This factor is an important indication of the deep changes in the state of the organism due to the extensive spread and multiplication of the *Treponema pallidum* in the organism and the changes in the organism's reactivity. The primary stage of syphilis is divided into two phases: (1) *primary, seronegative syphilis*—from the moment of the appearance of the hard chancre to the change in the serology tests from negative to positive, and (2) *primary, seropositive syphilis*—from the moment of the change in the serology tests from negative to positive to the appearance of the eruptions of the secondary stage.

An enlargement of all lymph nodes—specific polyadenitis— is observed 3-4 weeks after formation of the primary lesion. The enlarged lymph nodes are elongated, have a densely-elastic consistency and a smooth surface, and are painful. They do not adhere to each other or to the surrounding tissues and the skin, and are much smaller than those of the concurrent bubo in the same patient.

At the end of the primary stage of syphilis the multiplication of the *Treponema pallidum* reaches its maximum and the latter penetrates into the blood en masse.

During this period, usually 3-7 days before appearance of the eruption of the secondary stage, some patients exhibit prodromal symptoms, namely, weakness, indisposition, fatigability, irritability, headaches, dizziness, pains in the bones, joints and muscles, lack of appetite, insomnia and anemia. In some patients the prodromal period is accompanied by a feverish state, usually of

a subfebrile character; sometimes, however, the temperature may rise to 39°C and higher, which frequently leads to diagnostic errors. Often the prodromes are absent or are but feebly marked, and the patients attach no importance to them.

In some patients with recent secondary syphilis no vestiges of the primary lesion can be found. This is sometimes due to the peculiar localisation of the chancre on the urethral mucosa, on the cervix or in the cervical canal. In other, rarer, cases the primary lesion is really absent because these patients become infected with syphilis as the result of direct penetration of the *Treponema pallidum* into the circulation. This form of inoculation takes place, for example, in transfusion of the blood of a syphilitic.

In such cases no hard chancre forms at the site of inoculation and no concurrent bubo develops. In these patients syphilis begins, as it were, with the secondary stage, i.e., multiple eruptions on the skin and mucous membranes. However, these eruptions develop within the same period of time as in the patients with the usual course of the disease (with the hard chancre and scleradenitis), i.e., 8-10 weeks after inoculation. The reason for it is that the adaptation of the *Treponema pallidum* to the new conditions of existence in the organism, on the one hand, and the reorganisation of the organism's reactivity, on the other hand, require a certain period of time.

Primary syphilis is diagnosed on the basis of the aforedescribed picture of the primary lesion, the presence of the *Treponema pallidum* in its discharge, and the characteristic enlargement and induration of the adjacent lymph nodes.

The primary lesion may be confused with other diseases, namely, chancroid, erosive balanoposthitis and herpes progenitalis. Chancroid is characterised by acute inflammatory phenomena, soft ulcers with eroded edges and purulent discharge, and pain. Chancroid is most commonly marked by multiple ulcers whose discharge contains the Ferrari-Petersen streptobacillus. In this disease the regional lymph nodes are affected in less than 50 per cent of the cases. The lymph nodes are painful, adhere to each other and to the skin above and often suppurate.

Erosive balanoposthitis is characterised by numerous bright-red, very superficial, often coalescing erosions of various sizes and forms. The patient complains of pain, burning and itching. Observance of cleanliness and application of nonirritating agents, usually physiologic solution, rapidly terminate the disease.

Herpes progenitalis is marked by appearance of erythema and small vesicles arranged in groups, the vesicles bursting and forming erosions. The disease often repeatedly relapses. Establishment of a correct diagnosis is aided by the presence of vesicles or fragments of their covers retained on the edges of the erosions.

The secondary stage of syphilis begins with the appearance of eruptions on the skin and mucous membranes usually 8-10 weeks after inoculation. These eruptions are characterised by multiplicity and great variety.

The eruptions of the secondary stage or *secondary syphilids* are divided into: (1) macular, (2) papular, (3) pustular, and (4) pigmented. Despite their clinical diversity, however, they have common characteristic features.

General Characteristics of the Secondary Stage Eruptions

1. All syphilids are characterised by the absence of phenomena of acute inflammation. These eruptions do not have a bright inflammatory colouring; they are of a faded red colour with a yellowish ("coppery") or bluish ("boiled ham") hue.

2. Subjective sensations are, as a rule, absent.

3. An infiltrate in the derma forms the basis of secondary syphilids; even in untreated cases the infiltrate is soon resorbed without leaving a permanent trace on the skin or the mucous membranes. This explains the *benignancy* of the eruptions of the secondary stage. Certain pustular syphilids which leave scars are the only exception to the rule.

4. The infiltrate of the eruptions of the secondary stage always contains a large number of the *Treponema pallidum*. If the continuity of the epidermis within the limits of the eruption lesion is disrupted, large numbers of the *Treponema pallidum* find their way to the surface of the lesion from the depth of the infiltrate.

5. Secondary syphilids are characterised by rather regular round contours and clearly-defined borders. Most secondary syphilids (except macular and pigmented) have a densely-elastic consistency.

6. After their appearance secondary syphilids scarcely enlarge and usually do not coalesce.

7. The serology tests in eruptions of the secondary stage of syphilis are nearly always positive. The Wassermann and flocculation tests are positive in 98-99 per cent of the patients with phenomena of secondary syphilis.

8. The eruptions of the secondary stage of syphilis disappear soon after the patient begins antisyphilitic treatment.

During the secondary stage of syphilis the eruptions do not all appear at once, but develop gradually (in spurts), and attain complete development 10-14 days after their appearance.

The clinical picture of the first eruptions of secondary syphilis (recent syphilis II) in many respects differs from that of recurrent eruptions (relapsing syphilis II). In recent secondary syphilis the eruptions are, as a rule, plentiful and the lesions are

distributed symmetrically, evenly and not in definite groups. The eruptions of relapsing secondary syphilis are scantier as regards the number of lesions, are distributed in groups and bunches, sometimes forming arches and rings.

In some patients, especially, debilitated and emaciated, most commonly in women, the eruptions of secondary syphilids are accompanied by a rise in temperature, in most cases subfebrile.

Roseola Syphilitica or Macular Syphilids

The syphilitic roseola or macular syphilitic eruption is one of the most common manifestations of secondary syphilis.

Upon its appearance the roseola looks like a pink or reddish, oval or irregularly shaped macule form several millimetres to a thumb-nail in diameter. Their borders are less clearly defined than those of other secondary syphilids (Figs. 112 and 121).

Usually roseolous macules do not coalesce; coalesced roseolas form only in cases of particularly copious eruptions. Roseolas cause no subjective sensations at all, and erupt not all at once, but in "spurts". The number of macules increases till the 10th-14th days, the macules appearing in unaffected as well as in affected parts of she skin. Roseolas most commonly localise on the lateral surfaces of the trunk, on the chest, abdomen, shoulders and forearms. Eruptions are sometimes also observed on the back, buttocks and thighs, rarely on the face and shanks, and hardly ever on the palms and soles of the feet. Newly appeared roseolas may undergo no perceptible changes for several days or even 2-3 weeks, after which they assume a purplish or brownish hue which gradually grows more intense; finally, the roseolas are transformed into pigmented macules which soon disappear without leaving a trace.

The surface of roseolas never scales. In the absence of treatment the reseolous eruption may last a total of 5-6 weeks and longer.

There is a certain difference between the roseolas of recent secondary and relapsing secondary syphilis. In cases of recent syphilis the roseolous eruption is usually plentiful and symmetrical, and the macules are not arranged in groups. A recurrent roseolous eruption is rarely plentiful, the number of macules is usually small. In a patient with relapsing secondary syphilis the roseolas are usually arranged in groups, bunches, arches and rings. In most cases the macules of recent secondary syphilis are not larger than a few millimetres in diameter. A relapsing roseola is, as a rule, larger, reaching the size of a large coin.

The syphilitic roseola must be distinguished from the macules of pityriasis rosea, pityriasis versicolor, the eruptions in toxi-

coderma and acute infectious eruption (typhoid fever, measles, scarlet fever, etc.).

The macules of pityriasis rosea are characterised by branny scaling and a yellowish colour in the centre. They gradually enlarge and are only rarely accompanied by itching.

Fig. 112. Syphilitic roseola

In pityriasis versicolor the macules of noninflammatory character are rarely pink or red, but are usually yellowish or brownish, and their surface scales. This disease is marked by a prolonged course, often of many years' duration. In toxicoderma the macules very often coalesce and in many cases cause itching; the lymph nodes are not enlarged, nor are there any other signs of secondary syphilis. An acute infectious eruption can be distinguished by its character (minute, pinpoint, red eruption against a pink background in scarlet fever, hemorrhagic hue of the eruption in relapsing fever, etc.) as well as other signs typical of these diseases.

Papular Syphilids or Syphilitic Papules

Syphilitic papules, like roseolas, are among the most common eruptions of the secondary stage and very widely vary in their clinical picture. Their variety is due to the individual properties of the organism, as well as their localisation and the external irritating influences.

There are several clinical forms of syphilitic papules. The most common ones are *lenticular* papules, which are flat, at first of a pink-red and then copper-red or ham-red colour. They are roundish and have clearly-defined borders and a rather dense consistency. Drawing a finger over the skin on which there is a papule gives one a clear sensation of a dense infiltrate clearly demarcated from the surrounding skin. The papules are of the size of a lentil.

The surface of the papules is smooth and somewhat shiny (Figs. 113, 114 and 122). Papules may localise on any part of the skin and mucous membranes. Like roseolas, they do not all appear at once, but develop gradually, in spurts, over a period of 10-14 days. After their appearance the papules enlarge but little and subsequently cease to enlarge. Usually lenticular papules do not coalesce. They begin to disappear several weeks after their appearance, acquiring an increasingly more pronounced brown hue and becoming surmounted by thin scales; their infiltrate is gradually resorbed.

The scaling begins in the centre of the papules and then extends to their periphery. Old papules exhibit scales in the form of "collars" surrounding them. After complete resorption of the infiltrate a lenticular papule leaves a brownish pigmented macule which in some cases persists for a rather long time. In patients with recent secondary syphilis the lenticular papules are distributed in the main uniformly and symmetrically and show no tendency to grouping. Contrariwise, recurrent lenticular papules are very often arranged in groups, rings, arches and garlands.

Nummular papules occur much more rarely than do lenticular papules. They have all the features of lenticular papules, but are larger, sometimes as large as a 10-25 cent coin. Usually there are few nummular papules and they are observed mainly in patients with relapsing secondary syphilis (Fig. 115).

Miliary papules may also resemble lenticular papules, but differ from them by their very small size—from that of pinpoint to that of a millet seed. They are roundish or conical and are often of a follicular character, i.e., each papule is formed about the ostium of a sebaceous and hair follicle. Miliary papules are observed in patients with relapsing secondary syphilis, most commonly in debilitated, emaciated people, not infrequently in tuberculous patients. They localise mainly on the trunk and are nearly always.
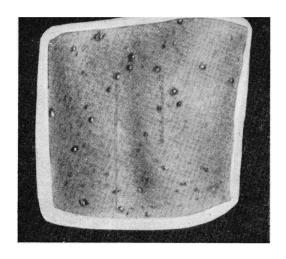
Fig. 113. Syphilitic lenticular papules (from the
Museum of the Central Institute of Skin and
Venereal Diseases)

Fig. 114. Lenticular papules
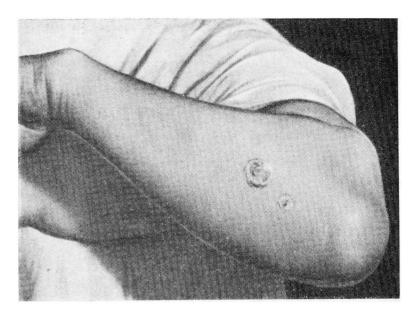
Fig. 115. Nummular syphilitic papules
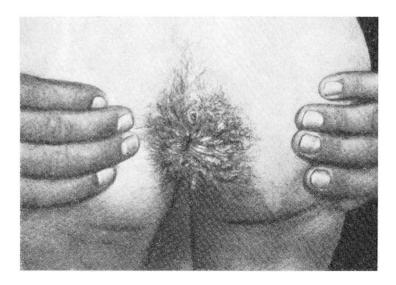
Fig. 116. Hypertrophic and exudative papules of the anus
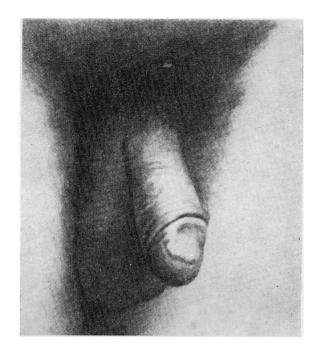
Fig. 117. Patchy papules on penis

Fig. 118. Exudative syphilitic papules
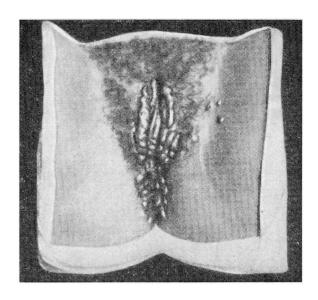
Fig. 119. Exudative syphilitic papules on external female genitalia (from the Museum of the Central Institute of Skin and Venereal Diseases)
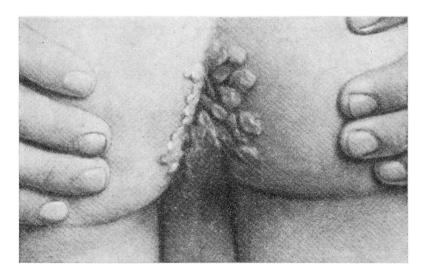
Fig. 120. Wide condylomas

Fig. 121. Syphilitic roseola (from multivolume manual of dermatology and venereology)

Fig. 122. Lenticular syphilitic papules (from multivolume manual of dermatology and venereology)

arranged in groups. When syphilitic papules are localised in skin folds, in the anal region or on the genitals they are irritated by friction, sweat and other excretions and enlarge. Such papules are called *hypertrophic* (Fig. 116). If the papules are located near each other, they coalesce as a result of peripheral growth and form continuous infiltrated patches with scalloped contours. Such eruptions are called *patchy papules* (Fig. 117). Very often irritation by friction and maceration by sweat and other excretions cause a swelling and maceration of the epidermis on the hypertrophic papules and patches, and the epidermis becomes opaque and whitish.

Intensified inflammatory phenomena cause a discharge of serous exudate from the depth of the papular infiltrate through the loosened, macerated epidermis; the discharge makes the surface of the papules moist. Such papules are called *moist* (Figs. 118 and 119). If the irritation by friction and sweat or other excretions does not cease, the macerated epidermis desquamates and the surface of the papules is changed to an erosion. The erosion has a smooth, red floor and a serous discharge; along the periphery it is surrounded by a border of papular infiltrate. These eruptions are referred to as *erosive papules*. In some cases a deeper necrosis develops in the papules, the latter becoming ulcerated and leaving scars. Large numbers of the *Treponema pallidum* are easily discovered in moist and erosive papules.

Protracted irritation may lead to coalescence of hypertrophic, moist and erosive papules into large patches. Such patches are considerably elevated above the level of the surrounding skin (up to 1-2 cm) and are characterised by very dense consistency; these patches are called *wide condylomas* (Fig. 120). Their surface may be moist or erosive. Wide condylomas heal slowly.

Moist and erosive papules and wide condylomas are particularly frequently localised in the intergluteal fold, around the anus and on the external genitalia. They are extremely contagious.

Syphilitic papules should be distinguished from nonsyphilitic papular eruptions. The lenticular, nummular and patchy syphilitic papules must not be confused with the papules and patches of psoriasis. Psoriatic papules and patches are usually pink-red (unlike the copper-red or ham-red syphilitic papules) and in most cases desquamate plentifully. All three symptoms—stearin macule, film and petechial hemorrhage—can be easily observed on psoriatic papules and patches, but are absent in syphilitic papules Lastly, most syphilitic patients exhibit other syphilitic eruptions (roseola, angina, etc.), polyadenitis, and positive Wassermann and flocculation tests. Miliary syphilitic papules resemble the papules of lichen ruber planus, but are pointed. The papules of lichen ruber planus are angular, flat, lustreless (waxy) and often umbilicated in the centre.

Erosive syphilitic papules on the genitals and in the anal region sometimes simulate lesions of pyodermas—folliculitis and impetigo. However, the erosive syphilitic papules have a dense infiltrate clearly demarcated from the surrounding tissues and develop from usual lenticular papules and not from pustules. The *Treponema pallidum* is easily found under the microscope in the discharge from erosive papules.

The diagnosis is facilitated by the presence of other eruptions of secondary syphilis on the skin and mucous membranes, and by positive Wassermann and flocculation tests.

Wide condylomas should be distinguished from acuminate condylomas, a disease closely related to ordinary warts. Acuminate condylomas are observed on the genitals as a result of irritation of the skin and mucous membranes by leukorrhea, pus and sweat. They are attached by a thin pedicle and consist of lobular outgrowths resembling cauliflower. Wide condylomas have no pedicles and lie on clearly demarcated densely-elastic infiltrate. The *Treponema pallidum* can easily be found in the discharge on their surface.

Pustular Syphilids

Pustular eruptions occur much less frequently in the secondary stage of syphilis. Pustular syphilids may be superficial and somewhat deeper. The former include impetiginous, acneform and varioliform syphilids. As the designations indicate, superficial pustular syphilids resemble vulgar impetigo, acne vulgaris and pustules of smallpox and chickenpox. However, all of them have the following very essential characteristic: a papule forms first and then rather rapidly develops into a pustule. That is why pustules are always surrounded by an areola of the papular infiltrate. Superficial pustular syphilids heal without leaving scars

Deep pustular syphilids include syphilitic ecthyma and rupia. In these forms of pustular syphilids the disintegration and suppuration are deeper and always involve the derma, for which reason pustules develop into ulcers surrounded by a border of dense infiltrate usually covered by a crust.

Syphilitic ecthyma and rupia resemble vulgar (streptococcal) ecthyma, but differ from it by the absence of acute inflammatory phenomena and the presence of a dense infiltrative areola Ecthyma and rupia possess marked peripheral growth, i.e., gradual increase in infiltrate and enlargement of the ulcers. Ecthyma and rupia heal slowly and always leave scars

Superficial pustular syphilids usually occur in recent secondary syphilis, ecthyma and rupia—mainly in relapsing syphilis. Pustular syphilids are a manifestation of the organism's lowered resistance and are usually observed in debilitated and emaciated patients and in alcoholics. Patients with pustular syphilids often

complain of headaches, a feverish state and pains in the bones and muscles. In such patients Wassermann and flocculation tests are often negative, which in the presence of active manifestations of secondary syphilis denote a weakened reactivity of the organism. Ecthyma and rupia are indications of a particularly unfavourable course of secondary syphilis.

Secondary Syphilids of the Mucous Membranes

Appearance of syphilitic eruptions on the mucous membranes is very often observed in the secondary stage of syphilis. These eruptions may be papular and macular. Macular eruptions most
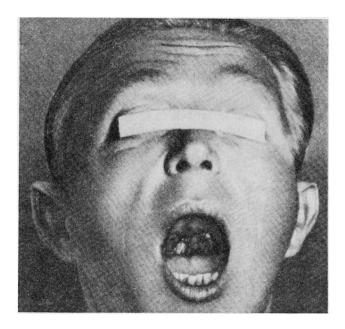
Fig. 123. Papular syphilitic angina

commonly occur in the oral cavity in the form of *erythematous syphilitic angina* which differs from common catarrhal angina by clearly defined borders of redness in the fauces, absence of edema, of pain on swallowing and of fever.

Papular eruptions are particularly frequently observed on the mucous membranes of female genitalia and anus, as well as in the oral cavity. Due to maceration these papules appear whitish. Continuous irritation of the papules (by saliva, food in the mouth, feces, friction in the anus, urine and discharges from the genitals)

causes them to grow peripherally and to coalesce. On the tonsils coalesced papules covered with a greyish-white membrane of macerated epithelium (papular syphilitic angina) sometimes resemble the picture of diphtheritic fauces. However, the slow development of papular angina, absence of pain on swallowing, the presence of other eruptions of secondary syphilis, polyadenitis, and the positive Wassermann test make it possible to establish a correct diagnosis.

The epithelium covering the papules on the mucous membranes easily desquamates and the papules become erosive or even ulcerative (Fig. 123).

Secondary syphilids of the mucous membranes are very contagious because the *Treponema pallidum* easily penetrates from the submucous infiltrate to the surface of the macules or papules through the macerated epithelium. That is why the surface of the syphilitic eruption always teems with the *Treponema pallidum*.

Pigmented Syphilid or Syphilitic Leukoderma

Syphilitic leukoderma develops 4-6 months after inoculation, i.e., it is one of the manifestations of recurrent secondary syphilis. It localises most commonly on the posterior and lateral surfaces of the neck. Sometimes it involves large areas of the skin, the region of the shoulder joints, the upper part of the back, the chest, etc.

The affected areas exhibit depigmented white macules surrounded by a border of yellowish-brown or light-brown hyperpigmentation. The macules are roundish, 2-3 mm in diameter and even larger (Fig. 124). The combination of the depigmented macules and hyperpigmented areas make the skin look like a net or lace. Syphlitic leukoderma is more frequently observed in women. The leukodermic macules may persist several months and sometimes even several years. These eruptions yield to antisyphilitic treatment slowly.

Alopecia Syphilitica

During the secondary stage of syphilis some patients exhibit diffuse or microfocal alopecia. In diffuse syphilitic alopecia the hair falls out all over the head and no separate bald patches are formed.

Microfocal alopecia usually develops 4-6 months after inoculation, i.e., at the time of the eruptions of relapsing syphilis. The picture of microfocal syphilitic alopecia is characteristic. Roundish or oval foci varying in size from that of a lentil to that of a 10-cent coin with thinned or even completely fallen-out hair appear on the hairy part of the head (Fig. 125). The skin in these foci presents the appearance of moth-eaten fur.

Fig. 125. Microfocal syphilitic alopecia (from the Museum of the Central Institute of Skin and Venereal Diseases)
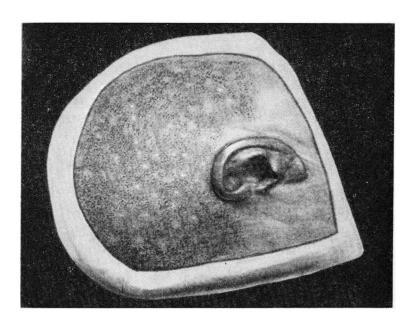
Fig. 124. Syphilitic leukoderma (from the Museum of the Central Institute of Skin and Venereal Diseases)

Microfocal syphilitic alopecia readily yields to treatment: several weeks after institution of the treatment the hair begins to grow back on the bald patches. Even without treatment the hair soon ceases to fall out both in diffuse and microfocal alopecia, new hair begins to grow and within a few months the hair is completely restored.

In most cases the eruptions of secondary syphilis are accompanied by an enlargement of all lymph nodes—syphilitic *polyadenitis*. In relapsing secondary syphilis polyadenitis may be more feebly marked, especially in relapses in the second or third year after inoculation and later.

In addition to the eruptions on the skin and mucous membranes, patients with secondary syphilis develop lesions in internal organs, the nervous system and bones.

Disturbances in a number of functions of the organism may be discovered by special examinations. However, these disturbances are not always clearly marked clinically. For example, it has been established by special studies that the hepatic function is disturbed in 70 per cent of the patients with secondary syphilis, although there are usually no clinical signs of this affection. Syphilitic meningitis is one of the rather commonly observed affections of the nervous system (in 10-30 per cent of the patients with secondary syphilis). In most cases, however, it shows no clinical symptoms (latent meningitis) and can be diagnosed only by examination of the cerebrospinal fluid. It is characteristic that secondary syphilitic affections of the internal organs, nervous system and bones for the most part run a benign course and soon pass off without leaving any permanent changes in the affected organs.

In most untreated cases the secondary stage of syphilis lasts about 3 years. The first eruption of the secondary stage—recent secondary syphilis—is followed by a latent period of syphilis—latent secondary syphilis which after a while (different in the different patients) is in its turn followed by new active manifestations of the secondary stage—relapsing secondary syphilis.

Latent secondary syphilis is usually characterised by a total absence of any symptoms that may warrant suspicion of syphilis in the patient. However, in many cases of latent secondary syphilis the Wassermann and flocculation tests are positive. In other cases the serology tests may also long remain negative. Nevertheless, in every case of latent secondary syphilis the syphilitic infection may at any moment become activated if the patient fails to take the necessary treatment.

Activation of the *Treponema pallidum* and diminution in the resistance of the patient's organism occur under the influence of various changes in the organism. These unfavourable changes may be due to harmful influences of the external environment (over-

Fig. 126. Grouped nodular syphilid (from multivolume manual of dermatology and venereology)

strain, poor hygienic conditions, inadequate diet, etc.), nervous shock and other disturbances in the activity of the central nervous system or other diseases, and intoxications.

The secondary stage of syphilis is of great social significance because of the prolonged course and the extreme contagiousness of the disease at this stage.

TERTIARY SYPHILIS

In most cases the tertiary stage of syphilis sets in 3-4 years after inoculation; this does not occur immediately after disappearance of the phenomena of the secondary stage, but after a rather long latent period of the disease.

The syphilitic infection may develop without the tertiary stage; not all patients go through this stage of the disease. Tertiary phenomena of syphilis are observed mainly in untreated or improperly treated patients. Modern antisyphilitic treatment serves as a reliable prevention of the tertiary stage of the disease. But even in the absence of treatment tertiary phenomena develop only in about 50 per cent of syphilis cases.

The onset of the tertiary stage of syphilis is favoured by a number of conditions which lower the resistance of the organism. These include chronic diseases, alcoholism and other intoxications, unfavourable living and working conditions, and various traumas. It is well known that tertiary syphilids of the skin most commonly develop at sites suffering frequent traumas—bruises, friction, etc. V. Tarnovsky observed that in patients with tertiary syphilis gummatous infiltrates soon form at cauterised sites. Gummatous infiltrates may develop at the sites of operative traumas in inadequately treated patients.

The patient's age also plays some part in the onset of the tertiary stage. Tertiary manifestations of syphilis are most commonly observed in children and old people.

Affections of the skin, mucous membranes and osseous system are usually observed during the tertiary stage; the internal organs and nervous system are also often affected. The clinical picture of tertiary syphilis is quite varied, yet the tertiary syphilids of the skin and mucous membranes have a number of characteristic features in common.

1. Tertiary syphilids are few and are usually localised on a circumscribed area of the skin and the mucosa.

2. Tertiary syphilids are characterised by the presence of a deep infiltrate in the derma or the subcutaneous adipose tissue. The infiltrate is of a densely-elastic consistency.

3. Tertiary syphilids develop slowly and run a chronic course with no phenomena of acute inflammation.

4. Tertiary syphilids run a malignant course—their infiltrate

usually disintegrates (often with formation of an ulcer) and leaves a scar.

5. Tertiary syphilids are characterised by a small number of the *Treponema pallidum* in the tissue of the infiltrate.

6. Tertiary syphilids develop without pain and other subjective sensations, or with very mild sensations.

7. Wassermann and flocculation tests are positive in about 75 per cent of the cases of tertiary syphilis.

8. Tertiary syphilids quickly heal with antisyphilitic treatment.

The manifestations of tertiary syphilis are hardly contagious because of the very few *Treponema pallidum* in the discharge from the ulcers of these patients. The *Treponema pallidum* is found with difficulty and in small numbers, and usually not on the surface of ulcers, but in the undisintegrated part of the infiltrate, for which reason cases of infection from patients with the tertiary stage of syphilis are observed extremely rarely.

There are two clinical forms of tertiary lesions in the skin — nodular and gummatous syphilids.

Nodular Syphilids

A nodular syphilid is a comparatively more superficial tertiary stage lesion in the skin with its infiltrate in the derma. The nodule appears as a flat or semispherical structure somewhat raised above the level of the skin. The contours of the nodule are roundish, the borders of the infiltrate demarcating it from the surrounding skin are clearly defined. The nodule varies in size from that of a hemp seed to that of a pea; it is indurated, copper-red, purplish-red or crimson-red and subsequently brownish (Fig. 126). It scarcely changes in size after its formation, although it may slightly enlarge for a short time after its appearance. It resolves either dry or by ulceration, but with necrosis in either case. In cases of "dry" resorption the nodule becomes flatter and brownish with lamellar scaling on its surface; it heals by leaving only cicatricial atrophy and temporary pigmentation. In cases of ulceration the disintegration of the infiltrate is more clearly defined and an ulcer is formed. Usually a closely-adhering dark crust forms in the centre of the nodule; removal of the crust reveals a round or oval ulcer with hard, round and, as it were, trimmed edges. The floor of the ulcer is covered with a necrotic yellowish membrane—the remains of the disintegrated infiltrate.

The ulcer heals leaving a scar which retains the form of the ulcer.

The scar often looks as though pressed into the skin. A thin border of depigmentation long persists around the scar. The entire development of the nodule ends within several months or even weeks.
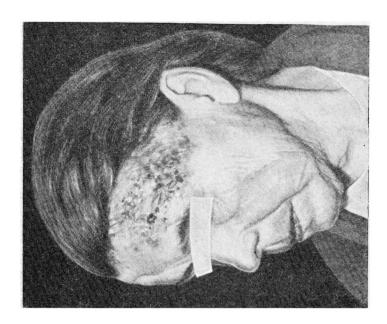
Fig. 128. Serpiginous nodular syphilid
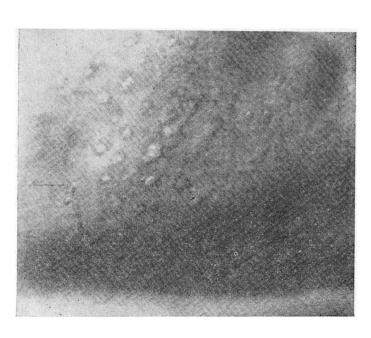
Fig. 127. Scars left by grouped nodular syphilid
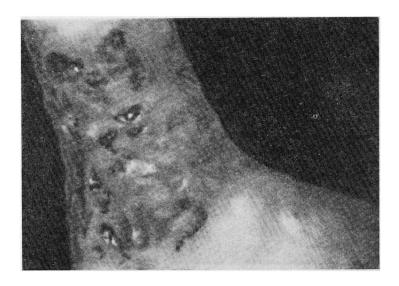
Fig. 129. Serpiginous nodular syphilid
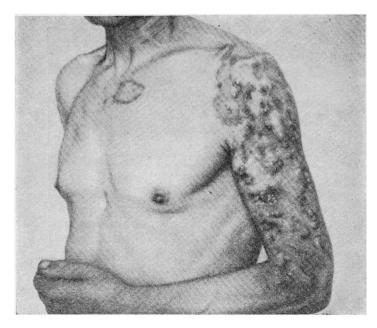
Fig. 130. Serpiginous nodular syphilid

Usually the nodules number 2 or 3 dozen, but in some cases there are single nodules; they localise for the most part on a circumscribed area of the skin. The following syphilids are distinguished according to the distribution of nodules: (1) grouped nodular syphilids, (2) serpiginous nodular syphilids, (3) areal nodular syphilids, and (4) micronodular syphilids.
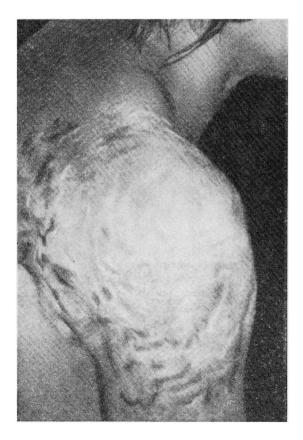
Fig. 131. Scar left by serpiginous nodular syphilid

In *grouped* syphilids the nodules are arranged in groups, bunches, sometimes in the form of an arch or crescent; they do not coalesce and heal by leaving scars separated by sections of healthy skin (Fig. 127).

In *serpiginous* or *creeping* nodular syphilids , as the nodules heal, new nodules are formed along the edges of the focus. An impression is created that the nodules are *creeping* in a certain direction, moving from the old sites to new areas. Some nodules

are so close to each other that they coalesce (Figs. 128-130). Ulceration of such coalesced nodules results in formation of irregularly shaped ulcers with scalloped edges. In serpiginous syphilids the nodules are often arranged in the form of arches or garlands, sometimes involving very large areas, for example, the entire shank, forearm, chest. Since the nodules are located very close to each other and are partly coalesced serpiginous syphilids leave continuous scars with scalloped edges and an uneven mosaic surface. The surface of the scars is uneven and has a mosaic pattern because in serpiginous syphilids the nodules do not usually coalesce completely, the infiltrates of the various nodules differ in extent and the ulcers are not of the same depth. The scars are particoloured; at the old sites they are depigmented and white, and at new sites they are brownish (Fig. 131).

In areal *ncdular* syphilids the various nodules completely coalesce into continuous infiltrated patches. This variety of nodular syphilids occurs rarely, as do also micronodular syphilids; the nodules of the latter are as small as millet or hemp seeds and their infiltrate is in the upper layer of the derma. The small nodules are for the most part resorbed dry and rarely become ulcerated.

A nodular syphilid may localise on any part of the skin, but does so particularly frequently on the extensor surfaces of the limbs, on the forehead and nose, in the region of the scapulae and on the small of the back. Sometimes these eruptions somewhat resemble lupus. However, lupus develops much more slowly and often occurs in childhood or youth. In lupus the nodules have a soft consistency and leave smooth, thin scars. Lupus scars are also characterised by the fact that they often become surmounted by new nodules, whereas no new nodules ever form on scars left by nodular syphilids. Nodular syphilids are observed in about 40 per cent of the patients with tertiary syphilis.

Gummatous Syphilids

A gammatous syphilid or syphilitic gumma is an infiltrated node, at first not larger than a bean, located deep in the subcutaneous tissue. The node is spherical, indurated and painless. By gradually increasing in size it may become as large as a walnut, a chicken egg and even larger, and begins to rise above the level of the surrounding skin. As the gumma enlarges it involves the derma; the skin above the gumma adheres to the infiltrate and becomes brown-red or purplish-red. The centre of the node soon softens, the skin thins out and breaks open with a small orifice, discharging a scant amount of gluey pus resembling gum arabic (hence, the term "gumma"). The orifice rapidly enlarges and soon a round ulcer with indurated, rolled edges steeply sloping
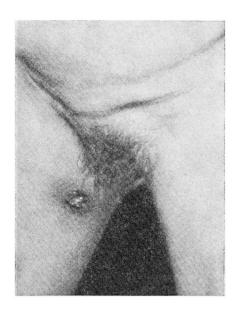
Fig. 132. Open syphilitic gumma
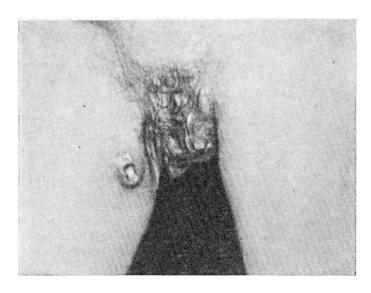
Fig. 133. The same gumma after detachment of the gummatous core

to the floor of the ulcer forms (Fig. 132). During the first days after formation of the ulcer its floor is covered with a closely adhering dirty-yellowish mass—necrotic infiltrate, the core of the gumma. In a few days the core detaches itself from the ulcer and leaves steep, as though trimmed edges and an uneven red floor covered with a purulent discharge.
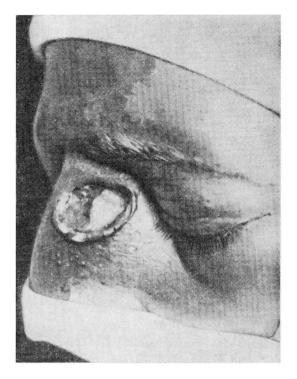
Fig. 134. Gummatous ulcer (from the Museum of the Central Institute of Skin and Venereal Diseases)

The ulcer is painless or but barely painful. The pain increases in intensity when the gummatous infiltrate involves nerves and the periosteum, and in cases of concurrent secondary infection. The gummatous ulcer slowly fills with granulations and cicatrises (Figs. 133-136). The development takes several months. Gummas may localise on any part of the skin. They form particularly frequently on the anterior surface of the shank, on the forehead, forearms and thighs, and in the region of the sternum. Gummas are never numerous. Patients most commonly have 1-2-3 gummas and but rarely up to 5-6 gummas. In cases of several gummas the

latter most frequently arrange themselves on one circumscribed portion of the skin; at the same time they may coalesce and form large, irregularly-shaped ulcers with scalloped edges.

Softening of the infiltrate and formation of an ulcer is the most common way of development of gummas. After healing the gummatous ulcers leave roundish scars, sometimes with scalloped edges. "Dry" resorption of the infiltrate is observed less frequently.

Fig. 135. Gumma of the shank (from the Museum of the Central Institute of Skin and Venereal Diseases)
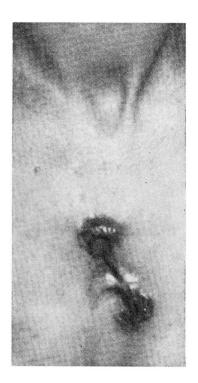
Fig. 136. Cicatrising gummatous ulcer

If the infiltrate involves deeper tissues — fasciae, muscles or bones — an umbilicated, sometimes stellate, scar is formed. An areola of hyperpigmentation often long persists around the scar (Fig. 137a). Nodular syphilids and gummas very often affect the mucosa of the mouth, nose and fauces. Tertiary syphilids are particularly frequently observed in the oral cavity, localised on the soft palate and the uvula. Affection of the soft palate imparts a nasal tone to the voice. Disintegration and ulceration of tertiary syphilids of the mucous membranes often lead to extensive

Fig. 137a. Scar left by syphilitic gumma of the shoulder
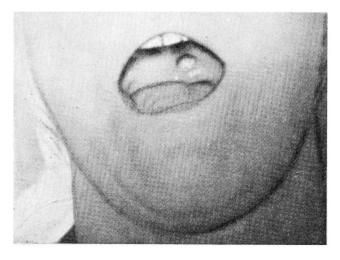
Fig. 137b. Gummatous ulcer of the soft palate

destruction, deformation of the remaining tissues and disfigurement (Fig. 137b). For example, affection of the soft palate not infrequently ends in sharp deformation and destruction of the velum palatinum and the uvula.

Syphilitic gummas must be distinguished from scrofuloderma and trophic ulcers. The scrofuloderma ulcers are irregularly shaped and soft. Their edges are loosened and hang over the ulcer floor; the floor readily bleeds and the discharge is thin and purulent. Scrofuloderma ulcers most commonly localise on the neck, and the scars they leave are characterised by skin "papillae" and "bridges". Trophic ulcers are usually distributed on the shanks and are most frequently associated with disturbances in the blood circulation due to varicose veins. They are mostly irregularly-shaped and are surrounded by infiltrated and edematous skin. They are chronic and often recur.

Tertiary Syphilids of the Bones

Affections of the osseous system are observed in 20-30 per cent of the patients with tertiary syphilis. Gummas of the bones usually derive from the periosteum—gummatous osteoperiostitides.
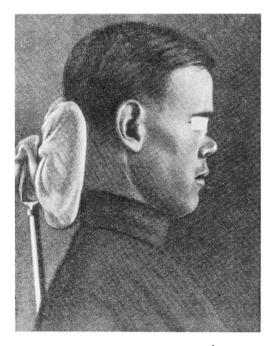
Fig. 138. Saddle nose (from P. Grigoriev)

Gummatous osteomyelitides are also observed. Gummas most commonly form in the tibias, although the bones of the nose, skull, forearm and the clavicles are also quite frequently affected. The gummas deriving from bones in most cases involve the overlying subcutaneous tissue and skin.

Tertiary osteoperiostitides are in many cases accompanied by nocturnal pains in the affected bones. These affections do not always result in disintegration of the infiltrate and ulceration. Often, especially in cases of osteoperiostitis of the tibia, connective tissue cells begin to proliferate in the infiltrate and the latter gradually becomes ossified. As a result the bone thickens and its surface becomes uneven.

Gummas of the nasal bones lead to considerable destruction and deformations of the nose. Due to the destruction of the bony base of the nose the dorsum nasi most commonly caves in and the characteristic saddle nose is formed (Fig. 138).

Owing to the lack of pain, which is characteristic of tertiary syphilids, patients usually apply for medical aid late, i.e., after irreparable destruction of the nasal bones and irremediable deformation of the nose. The deformation of the nose may be somewhat corrected only by a plastic operation which can be performed only after the end of the patient's antisyphilitic treatment.

Visceral Tertiary Syphilis

Affections of internal organs during the tertiary stage of syphilis are not rare. Syphilitic gummas may develop in any organ. Formation of a gumma in a vitally important organs (the brain, liver, etc.) and its subsequent disintegration lead to serious disturbances in the functions of the affected organ and even death. In addition to typical gummas, multiple miliary (small) gummas and diffuse infiltrates are also observed in the internal organs.

The most common manifestation of visceral tertiary syphilis is syphilitic aortitis which develops late—10-20 years after inoculation. A syphilitic infiltrate forms in the wall of the aorta, mainly in its muscular tissue. The patient sometimes has pain in the heart, irradiating to the shoulders and arms, dyspnea, tachycardia and a sense of substernal pressure.

In other cases there are no subjective sensations for a long time. X-ray pictures show an expanded shadow of the aorta.

The syphilitic process most commonly affects the ascending aorta. Owing to the formation of scar tissue at the site of the syphilitic infiltrate many patients affected with syphilitic aortitis develop an aortic aneurysm, i.e., a sharp dilatation and protrusion of the aortic wall. Aortic aneurysm is a severe disease involving cardiac dysfunction and circulatory disturbances; it often ends

in rupture of the thinned wall of the aneurysm with a fatal hemorrhage.

In addition to aortitis, syphilitic affections of other large arteries are also quite often observed during the tertiary stage.

Tertiary syphilis of the liver may manifest itself in the form of various gummas, multiple small (miliary) gummas and hepatitides with affections of the glandular, as well as the interstitial connective tissue.

Gummas of the liver form, for the most part, under its fibrous capsule. If the gumma is localised in the region of the porta hepatis through which the portal vein, hepatic artery and bile duct enter the liver, it may compress them and cause serious disorders of hepatic and abdominal circulation, ascites and mechanical jaundice. The patient complains of pain in the region of the liver, headaches, irritability, weakness, sometimes chills and elevated temperature. In cases of multiple gummas the surface of the liver may appear uneven and lobular to the touch.

In miliary gummas the liver is enlarged, indurated and painful; attacks of pain in the region of the liver and fever may be observed. Subsequently the gummatous infiltrate is replaced by scar tissue which may compress the hepatic blood vessels and cause hemostasis in the abdominal organs. The liver decreases in size, becomes compact and painless.

Tertiary syphilitic hepatitis with affection of the glandular or interstitial tissue is accompanied by enlargement of the liver, jaundice, sometimes pains in the region of the liver, general indisposition, itching and ascites. Later the liver decreases in size and becomes indurated; sometimes cirrhosis of the liver develops. The disease may terminate in death due to hepatic insufficiency.

Affection of the kidneys may assume the form of nephrosis, nephritis and gummas of the kidneys.

Gummas of the skin, mucous membranes, bones and internal organs are amenable to antisyphilitic treatment. Even large gummas are resorbed "dry" without ulcerating comparatively soon after the beginning of the treatment. This amenability of tertiary syphilids to the action of antisyphilitic preparations is sometimes used for diagnostic purposes. In dubious cases, when the clinical picture of the disease resembles tertiary syphilis, but there is no incontestable proof of the syphilitic nature of the disease and the serology tests are negative, the patients are administered test treatment. In such cases patients are prescribed antisyphilitic agents (most commonly potassium iodide and bismuth preparations) and the results of the treatment are watched. Rapid success of such treatment denotes a syphilitic origin of the disease. Test treatment is administered only by prescription of a physician.

The duration of the tertiary stage of syphilis varies very widely and depends on the individual state of the organism and the

patient's externall conditions. If the patient is given antisyphilitic treatment, the tertiary phenomena rapidly disappear and, as a rule, never recur. But, if the patient is not treated or treated poorly, the tertiary stage may last a long time.

The nodules or gummas appearing in the beginning of the tertiary stage may within a certain period of time heal also without treatment, but the disease enters a latent state—latent tertiary syphilis. After a certain latent period active tertiary manifestations of syphilis may develop again. In the absence of treatment active manifestations of the tertiary stage of syphilis may alternate with periods of the latent state of the syphilitic infection for several years.

Neurosyphilis

Affections of the nervous system are observed in all stages of syphilis. It is believed that at the end of the primary stage the *Treponema pallidum* penetrates to the central nervous system in all cases, but only a minority of patients exhibit symptoms of affection of the central nervous system at this time.

Syphilitic affections of the nervous system observed in patients for the most part during the first 5 years after inoculation are referred to as early neurosyphilis. The affections are characterised by lesions of a chronic inflammatory character in the meninges and cerebral vessels. Syphilitic meningitides are most commonly observed during the secondary stage. In most patients they manifest themselves only in the form of changes in the cerebrospinal fluid, which denote the presence of an inflammatory process in the meninges. In the minority of patients with early neurosyphilis syphilitic meningitis is marked by headaches, dizzines, tinnitus, nausea, vomiting, insomnia, excitement and impairment of vision. Neuralgias, polyneuritides and disturbances in cerebral circulation are observed. Syphilitic affections of the nervous system during the secondary stage are characterised by comparative benignancy and a tendency to pass without leaving any permanent changes (even without treatment). In some patients, however, in the absence of treatment early neurosyphilis develops into late syphilitic affections.

Late neurosyphilis which develops 5-10-15 years after inoculation with syphilis includes the affections of the cerebral vessels and meninges of the tertiary stage of syphilis and the so-called parenchymatous neurosyphilis. The most common affections of the vessels and meninges in late neurosyphilis are affections of the cerebral arteries (arteritides), gummatous meningitis and cerebral gummas. All these forms of late neurosyphilis are marked by an unfavourable course and a tendency to disintegration with considerable disturbances in the functions of the central nervous system. These affections are characterised by intense headaches,

pareses and paralyses, convulsive seizures, and cerebral hemorrhages. A lethal outcome is possible, especially if the disintegrating gumma is localised in a vitally important part of the brain, and in cerebral arteritides. Parenchymatous neurosyphilis is characterised by an even severer course and includes tabes dorsalis and general paresis. These forms develop 8-15 years after inoculation. By their clinical picture and course tabes dorsalis and general paresis differ from the other forms of neurosyphilis. They are marked by sharply reduced resistance of the organism to the syphilitic infection. The term "parenchymatous neurosyphilis" emphasises that the pathologic process operates not in the cerebral vessels and meninges, but directly in the nervous tissue of the spinal cord and brain. Large numbers of the *Treponema pallidum* are found in the affected tissue of the central nervous system (posterior columns of the spinal cord in tabes dorsalis and the cerebral cortex in general paresis). However, the response inflammatory changes in the tissues are very feebly marked.

Signs of degeneration and necrosis of tissues prevail in the focus of affection.

The affection of the spinal cord in tabes dorsalis is accompanied by intense shooting pains along the course of various nerves, girdle pains, changes in the pupils (constriction, different sizes of the pupils of the right and left eyes, absence of reaction to light, irregular form of the pupils), disorders of the gait and other movements, atrophy of the optic nerves, and impotence. Severe cases are marked by disturbances in urination and defecation, paralyses, and poorly healing trophic ulcers of the lower limbs. The course of the disease is protracted, chronic.

General paresis is a mental disease. The mental disturbances may manifest themselves in excitement with delusions, as well as in depression and progressive feeble-mindedness. In addition to disturbances in higher nervous activity, pupillary changes, cerebral hemorrhages and paralyses are observed. The disease ends within 2-3-5 years in death with phenomena of extreme feeble-mindedness, paralyses and profound general degeneration (paralytic marasmus). General paresis and tabes dorsalis develop, as a rule, in patients untreated during the early stages of syphilis or treated insufficiently and irregularly.

In the Soviet Union parenchymatous neurosyphilis is a rare occurrence.

Comparatively recently these diseases were considered incurable. Today it is possible to arrest the progress of the disease and often considerably to improve the patient's general condition and restore his working capacity.

That syphilis could be transmitted to the offspring was known as early as the end of the 15th century, but wrong conceptions concerning infection of the fetus by the father through the male germ cell —the spermatozoon—long prevailed in science. Infection of the fetus by the mother was considered possible also only through the female germ cell.

It was later established that congenital syphilis is transmitted only through the placenta by the syphilitic mother.

The *Treponema pallidum* penetrates into the tissues of the fetus after 4 months of pregnancy, when the placenta has already formed. A healthy placenta is believed to be impenetrable to microbes, including the *Treponema pallidum*. But having penetrated into the placenta the *Treponema pallidum* produces a number of pathologic changes in it with the result that the placenta becomes penetrable to the causative agent of syphilis. The *Treponema pallidum* penetrates from the placenta into the tissues of the fetus with the blood through the umbilical veins or through the lymphatics of the umbilical cord.

However, a syphilitic mother does not always give birth to a child affected with congenitạl syphilis.

Syphilis is most commonly transmitted to the offspring during pregnancy in the first 3 years after inoculation of the mother (M. Raits). Particularly dangerous in this respect is a pregnancy during the first year after inoculation of the woman with syphilis. However, there have been many cases of syphilitic children born of women inoculated many years before the particular pregnancy.

The pregnancy of a syphilitic woman may end in:

(1) miscarriage in the 5th or 6th month with syphilitic changes in the organs of the fetus;

(2) premature delivery; stillborn child also with syphilitic changes in the organs;

(3) premature delivery; living but nonviable child with severe symptoms of congenital syphilis dying during the first days after birth;

(4) birth of a full term child with manifestations of congenital syphilis or a child born apparently healthy with signs of syphilis appearing during the first months of life;

(5) prematurely delivered or full term child without clear signs of syphilis, the signs of congenital syphilis developing later;

(6) birth of a healthy child.

The more recent the syphilitic infection of the mother, the graver its influence on the results of the pregnancy. If the inoculation of the mother occurred shortly before conception or soon afterwards, the pregnancy usually ends in a miscarriage or pre-

mature delivery of a dead child. Pregnancy following inoculation at a somewhat later period may end in the birth of a premature and nonviable child or a full term child with symptoms of congenital syphilis. Pregnancy started still later after inoculation often ends in the birth of a child without symptoms of congenital syphilis, but some time later, in some cases several years after birth, these symptoms appear just the same. In some cases the pregnancy started a long time after inoculation may end in the birth of a healthy child. Thus in the course of time the influence of the mother's syphilis on the results of the pregnancy seems to diminish. It should be remembered, however, that, in addition to the duration of the disease, the results of the pregnancy of a syphilitic mother are influenced by a number of factors, namely, reduced resistance of the organism and various internal diseases and intoxications which increase the danger of the child's inoculation. The transmission of syphilis from mother to child and the severity of the manifestations of congenital syphilis are depended on condition of the mother. If the syphilitic mother is treated adequately and regularly, her pregnancy, as a rule, ends in the birth of a healthy child. Unfinished and inadequate treatment is always fraught with the danger of unfavourable results of the pregnancy, i.e., miscarriage, stillbirth and birth of a child with congenital syphilis.

According to M. Raits, only 11 per cent of healthy children are born of untreated syphilitic mothers, while adequately and regularly treated syphilitic women give birth to healthy children in 97 per cent of the cases (M. Khoroshin).

There is a radical difference between the development of congenital and acquired syphilis. In cases of inoculation through the placenta the *Treponema pallidum* very soon invades all the organs and tissues of the fetus. Since the reactivity of the organism of the fetus is low and its immune processes are very feeble, the *Treponema pallidum* finds itself under conditions which greatly favour its existence and multiplication. The organism of the fetus turns out to be almost defenseless against the mass multiplication of the causative agent invading all its organs and tissues. The special conditions under which syphilis develops in cases of placental inoculation lead to characteristic manifestations of congenital syphilis, which in many respects differ from those of acquired syphilis.

The following manifestations of congenital syphilis are distinguished: (1) syphilis of the fetus, (2) congenital syphilis of infants, (3) congenital syphilis of early childhood, and (4) late congenital syphilis.

Syphilis of the fetus. In miscarriages symptoms of syphilis are discovered in the fetus between the 4th and 6th months. The fetus is destroyed as the result of the mass multiplication of the

Treponema pallidum in all its organs and of the disturbances in its nutrition caused by pathologic changes in the placenta.

In congenital syphilis the skin of the fetus is for the most part macerated, whitish and in some places detached in flaps. The greatest changes occur in the internal organs of the fetus. The liver is enlarged, indurated and brownish, its tissue teeming with the *Treponema pallidum* and containing inflammatory cellular infiltrate. The spleen is also enlarged and indurated. The lungs present a picture of *pneumonia alba*. The pulmonary tissue appears indurated, contains little air and is yellowish or white-pink. In the focus of affection the pulmonary alveoli are filled with degenerated cells of pulmonary epithelium. A very characteristic feature is the affection of the long tubular bones—syphilitic *osteochondritis*. The essence of this process consists in disturbed ossification. The calcification border of tubular bones (the long bones of the limbs, sometimes the ribs) normally appears in section or when examined roentgenologically as a straight, even and light band up to 0.5 mm wide. Penetration of the *Treponema pallidum* into the metaphysis of tubular bones where ossification takes place leads to a disturbance in the process of ossification. The calcification band widens to 2 mm, becomes uneven and serrate due to resorption of already formed bone and development of granulation tissue.

Congenital syphilis of infants. The manifestations of congenital syphilis of infants usually appear during the first 2 months of the child's life. The child often comes into the world with symptoms of congenital syphilis. Congenital syphilis of infants rarely occurs after 4 months of life. The earlier the signs of congenital syphilis appear in the child, the severer its course.

Infants affected with congenital syphilis are nearly always debilitated and develop poorly. Their weight is below normal, they have little subcutaneous tissue, appear emaciated and often look like "little old people" because of their flabby skin covered with deep wrinkles. Such infants very often cry almost incessantly and "without any reason".

Congenital syphilis of infants is characterised by copious and various eruptions on the skin and mucous membranes, and lesions in the bones and internal organs.

Diffuse infiltration of the skin is one of the most common skin affections in congenital syphilis of infants and is observed in 60 per cent of the cases. The usual localisation is the palms, the soles of the feet, the face and buttocks; the other parts of the skin are affected less frequently. The most commonly affected parts of the face are the cheeks, lips, chin and forehead. Pink-red macules are the first to appear in the affected areas. The skin in these areas becomes thickened, indurated, tense and saturated-red. The macules have a smooth and shiny surface. Subsequently they become
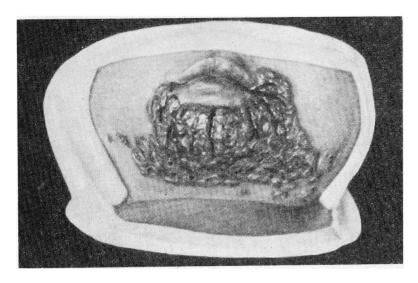
Fig. 139. Diffuse syphilitic infiltration of the skin (from
B. Pashkov)
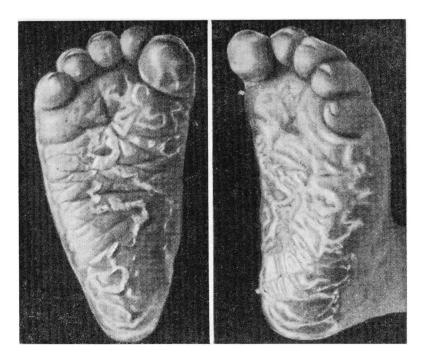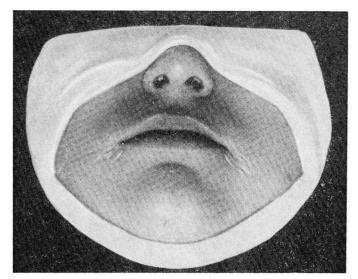
Fig. 140. Diffuse syphilitic infiltration of the skin (from
multivolume manual of dermatology and venereology)

a

b

Fig. 141*a* and *b*. Fournier's scars left by diffuse syphilitic
infiltration (from B. Pashkov)
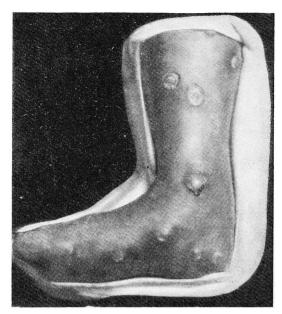
Fig. 142. Syphilitic pemphigus
(from M. Zheltakov)

Fig. 143. Diffuse infiltration and erosions (from multivolume
manual of dermatology and venereology)

brownish and their surface begins to desquamate (Figs. 139 and 140). Sometimes the foci of diffuse infiltration are transformed into an erosive, exudative surface, and become covered with crusts. The discharge from these foci contains large numbers of the *Treponema pallidum*. Diffuse infiltration of the skin often leads to

Fig. 144. Syphilitic rhinitis and diffuse infiltration of the skin and of the vermilion border with deep fissures (from A. Kartamyshev)

formation of deep fissures around the mouth. On healing the fissures leave characteristic lifelong white scars. The scars partly cover the vermilion border and adjacent portions of the skin and run transversely to the lips (Fournier's scars) (Figs. 141*a* and *b*). Diffuse infiltration usually appears several weeks after the birth of the child and is observed only in congenital syphilis of infants.

Syphilitic pemphigus is also peculiar only to infants with congenital syphilis and occurs only in about 2 per cent of the patients. It is a pustular syphilid indicating a severe malignant course of congenital syphilis. The child either comes into the world with eruptions of syphilitic pemphigus or the eruptions appear during the first days or weeks of the child's life. Blisters varying in size from that of a lentil to that of a pea and larger appear on the palms, the soles of the feet, less frequently on the

forearms and shanks, and still less frequently on other parts of the skin. Their walls are flabby; later they may become umbilicated in the centre. Then the content of the blisters becomes turbid and purulent, and the blisters develop into pustules. Along the periphery the pustules show a border of papular infiltrate. The pustules teem with the *Treponema pallidum* (Fig. 142).

Papular syphilids. Syphilitic papules are very often observed in infants affected with congenital syphilis. Owing to the tendency of the infants' skin to maceration and chafing the syphilitic papules not infrequently become exudative and erosive (Fig. 143).

Syphilitic roseolas are rarely observed in infants with congenital syphilis.

Syphilitic rhinitis. Syphilids of the mucous membranes are a frequent occurrence in congenital syphilis of infants, the affection of the mucosa of the nose in the form of syphilitic rhinitis being observed in 70 per cent of the cases (Fig. 144). Owing to a diffuse infiltrate in the submucous tissue of the nasal passages the lumens of the latter are greatly constricted. The thick sero-purulent discharge dries forming crusts which obstruct the nasal passages. This renders nasal breathing extremely difficult, the latter becoming sibilant, stertorous and stridulous. Sometimes the infant cannot breathe through the nose at all. The difficulties of nasal breathing hinder the infant from sucking the mother's breast; taking the breast greedily it begins to choke and gives up the breast with a cry. The infant tires before sucking the necessary amount of milk. Systematic undereating causes emaciation of the infant. The infant exhibits syphilitic rhinitis at its very birth or develops it during the first month of its life. Syphilitic rhinitis runs a chronic, stubborn course and yields to antisyphilitic treatment slowly.

Syphilitic rhinitis may be accompanied by ulceration of the infiltrated mucous membranes, involving the nasal bones and nasal septum in the process. As a result the nasal septum is often perforated, the dorsum nasi caves in and the nose becomes saddle-shaped.

Syphilitic osteochondritis. About 85 per cent of the infants affected with congenital syphilis have affections of the long tubular bones of the limbs; the essence of this affection was described above (see Syphilis of the Fetus). In most cases osteochondritis can be identified only through an X-ray picture of the bones. The far-advanced changes in the bones in osteochondritis sometimes lead to complete or almost complete separation of the epiphyses from the diaphyses with the resultant picture of Parrot's disease (syphilitic pseudoparalysis) in which the child holds the affected arm extended and pressed against the trunk and the affected leg flexed in the knee and drawn up to the abdomen. The child performs no active movements with the affected limbs (Fig. 145). At

attempts to change the position of the affected limb the child cries
bitterly with sharp pain. Children with osteochondritis and, espe-
cially, Parrot's disease must be treated with particular care be-
cause of the danger of pathologic fractures.
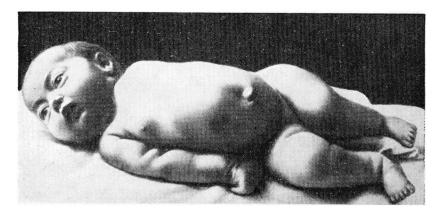
Fig. 145. Parrot's disease (syphilitic pseudoparalysis) (from
multivolume manual of dermatology and venereology)

Syphilitic osteochondritis is often observed at the birth of the
child or it develops during the first 3 months of the child's
life.

In addition to osteochondritis of the tubular bones, other affec-
tions of the osseous system are also frequently observed in con-
genital syphilis of infants; these affections include periostitides
of the tubular and cranial bones, gummas of the bones and affec-
tions of the finger bones.

The internal organs of infants with congenital syphilis are very
frequently affected. Enlargement and induration of the liver and
spleen are the most common affections (in 80 per cent of the pa-
tients). Syphilitic boys often exhibit affection of the testes and
their adnexa. The affected testes are enlarged, indurated and
painless.

Moderate enlargement and induration of the lymph nodes—
micropolyadenitis—are very often observed.

In active manifestations of congenital syphilis of infants Was-
sermann and flocculation tests are nearly always positive.

Infants with congenital syphilis suffer from various gastroin-
testinal diseases, influenza and pneumonia much more frequently
and severely than do healthy children; these diseases run a more
protracted course, often relapse and produce various complica-
tions and higher mortality.

Before the introduction of penicillin into the therapy of syphilis a large number of infants with congenital syphilis died in the first year of life.

CONGENITAL SYPHILIS OF EARLY CHILDHOOD

Manifestations of congenital syphilis characteristic of infants are extremely rarely observed already at the end of the first year of life. From 1 to 4 years of age congenital syphilis is for the most part latent because during the first year of the disease the resistance of the child's organism gradually increases and the processes of immunity intensify. In syphilis of early childhood the *Treponema pallidum* is less active.

The clinical manifestations of congenital syphilis in early childhood consist predominantly in papular eruptions which develop mainly in the anal region in the form of exudative and erosive papules and wide condylomas (Fig. 146). Papules also appear on the external genitalia and, less frequently, on the oral and nasal mucosa. Periostitides of the long tubular bones of the limbs, and diseases of the choroid and retina often develop.

The syphilitic infection unfavourably affects the general development of children. Patients with congenital syphilis of early childhood are often mentally retarded and physically underdeveloped.

In the absence of active manifestations of the disease in patients with congenital syphilis of infants or early childhood the condition is referred to as *congenital latent syphilis*.

Late congenital syphilis. The "calm" which characterises the course of congenital syphilis of early childhood is followed by a new period of active manifestations of syphilis—late congenital syphilis. These include the manifestations of congenital syphilis after 4 years of age and up to the time of sexual maturity.

Active manifestations of late congenital syphilis are more frequently observed at a later age—20-25 years and later. However, in a large majority of patients active manifestations of late congenital syphilis appear from 6-7 to 14 years of age.

Symptoms of the tertiary stage of acquired syphilis prevail in late congenital syphilis (Fig. 147). But in addition to the nodular and gummatous syphilids of the skin and mucous membranes, and the gummas of the bones and internal organs, this stage is characterised by a number of phenomena peculiar only to late congenital syphilis. These phenomena include Hutchinson's triad and the saber-shaped shank.

Hutchinson's triad consists of interstitial keratitis, eighth nerve deafness and notched teeth (Hutchinson's teeth).

Interstitial keratitis is a diffuse inflammation of the cornea. The disease manifests itself in the form of hyperemia and opacity
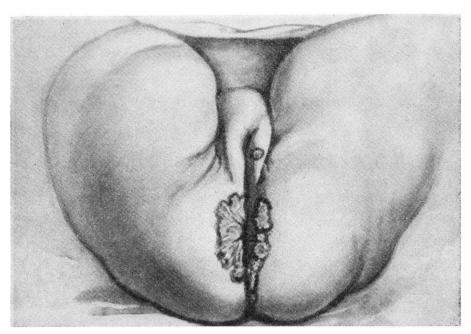
Fig. 146. Hypertrophic papules in syphilis of early childhood
(from multivolume manual of dermatology and venereology)
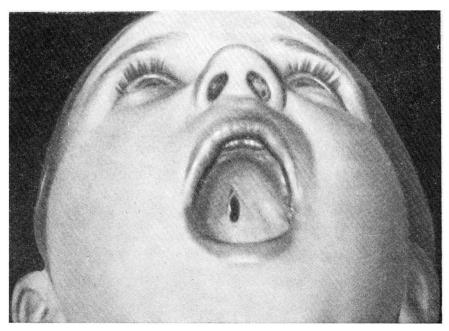
Fig. 147. Gummatous ulcer of the hard palate (from multivolume
manual of dermatology and venereology)

of the cornea, intense photophobia, spasm of the eyelid muscles, lacrimation and diminution in visual acuity. Usually one eye is affected first, but within a few months the affection also extends to the other eye.

Interstitial keratitis is observed in nearly 50 per cent of the patients with late congenital syphilis. It runs a chronic course and ends in a permanent opacity of the cornea and diminution in visual acuity, in some cases—in total blindness.

Eighth nerve deafness or *syphilitic labyrinthitis* develops rapidly and usually affects both ears. The patient suddenly develops tinnitus, sharp impairment of hearing, often total deafness, and sometimes dizziness. The disease is scarcely amenable to antisyphilitic treatment.

Hutchinson's teeth are a characteristic change in the form of the two middle incisors which look like chisels (wide base and a narrowing towards the free edge), are often skewed and have a semilunar notch along their free edge (Fig. 148). Hutchinson's teeth are observed in about one of every six patients with late congenital syphilis.

In the Soviet Union a full Hutchinson's triad is very rarely observed. The presence of even one member of the triad is therefore important for diagnosing late congenital syphilis.

Saber-shaped shanks are observed in late congenital syphilis quite often. The shanks are curved and look like sabers convex anteriorly (Fig. 149). The curvature of the shanks is due to their improper growth caused by an attack of osteochondritis in infancy. The anterior surface of the tibia is thickened and uneven; its crest is smoothed out. These changes are the result of periostitis with subsequent ossification of the infiltrate. In late congenital syphilis nodular and gummatous syphilids often develop, while active manifestations of the disease alternate with latent intervals. Late congenital syphilis without active manifestations is called *latent congenital syphilis*.

Patients with late congenital syphilis often have a number of changes, mainly in the bones and teeth, which are associated with the toxic effects of the metabolites and waste products of the *Treponema pallidum* on the child's growing organism, these metabolites and products leading to improper development of various organs and tissues. These developmental defects are referred to as *syphilitic dystrophies*. They include the thickening of the sternal end of the right clavicle, congenital absence of the xiphoid process, shortening of the little fingers which do not reach the bases of the terminal phalanges of the third fingers, deformations of the skull, high and narrow hard palate, irregular shapes and positions of the teeth, and certain other dystrophies.

However, the presence of these dystrophies cannot serve as absolute proof that the patient has syphilis and does not warrant
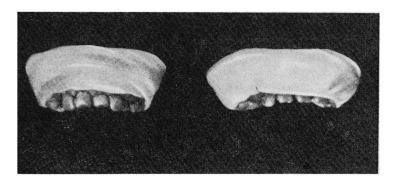
Fig. 148. Hutchinson's teeth (from the Museum of the Central
Institute of Skin and Venereal Diseases)
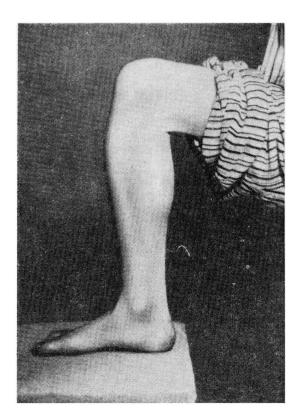
Fig. 149. Saber-shaped shank

a diagnosis of late congenital syphilis because such dystrophies may be caused not only by syphilis. In such cases it is necessary carefully to examine the patient and the other members of his family.

As in acquired syphilis, affections of the nervous system are also quite frequently observed in late congenital syphilis.

Prevention of Congenital Syphilis

Congenital syphilis exerts a profound adverse influence on the child's physical and mental development and leads to appearance of inferior progeny. Children affected with congenital syphilis more readily contract children's diseases, these diseases run a severer course and result in higher mortality. Hence the social significance of congenital syphilis. It goes without saying that prevention of congenital syphilis is one of the principal objectives of the control of venereal diseases. It is based on the following measures:

1. Proper and regular treatment of every syphilitic woman of child-bearing age.

2. Preventive treatment of every pregnant woman formerly affected with syphilis. Preventive treatment during pregnancy is administered even in cases where the women were adequately treated and finished their treatment before pregnancy.

3. Preventive examination of all pregnant women aimed at discovering syphilis, for which purpose every pregnant woman must be given Wassermann and flocculation tests. The tests must be performed twice in the course of pregnancy—in the first half and in the 6th or 7th month.

The examinations of pregnant women are not confined to serology tests. Pregnant women must be given a thorough clinical examination with special attention to the condition of the skin, mucous membranes, bones and lymph nodes.

It is necessary to obtain a detailed anamnesis from every pregnant woman, to ascertain the diseases she has survived and the state of health of her husband and children.

An exact anamnesis and careful clinical examination often make it possible to establish a past syphilitic infection in the pregnant woman even when the serology tests prove negative. The pregnant women are examined in the medical institutions where they are registered (maternity health centres, hospitals, physician assistant's or midwife's stations).

4. Preventive treatment of all children born of syphilitic untreated or inadequately treated mothers.

The planned control of congenital syphilis in the Soviet Union has led to a sharp reduction in the incidence of the disease in the country.

In 25 years (1925-1950) the number of congenital syphilis patients decreased scores of times and today this form of syphilis is a rare occurrence. For example, only 15 cases of early congenital syphilis were recorded in the Russian Federation in 1960.

The American venereologist Clark reports that 16,000 children are annually born with congenital syphilis in the U.S.A. According to Zappert, 3.3 per cent of all the newborn in the U.S.A. are affected with congenital syphilis. It should be noted that Clark's and Zappert's figures may be lower than the actual figures for lack of accurate records of venereal patients in the U.S.A.

SEROLOGY TESTS IN SYPHILIS

In the presence of active manifestations of syphilis the disease is diagnosed on the basis of clinical signs. The clinical diagnosis is confirmed by laboratory data, for which purpose the discharge from primary lesions or from exudative and erosive papules is examined for the *Treponema pallidum*, serology tests are made and the cerebrospinal fluid is examined. In some cases the diagnosis of syphilis may be established on the basis of the consequences of active manifestations of syphilis suffered by the patient in the past. These are, for example, typical scars left by nodular syphilis, Fournier's scars on the lips in congenital syphilis, Hutchinson's teeth, saber-shaped shanks, etc.

Serology tests play a particularly important part in the diagnosis of syphilis. During the active periods of the disease serology tests serve to confirm the diagnosis of syphilis, which is extremely important in cases with an unclear and dubious picture. The role of serology tests is still more important in establishing the diagnosis of latent syphilis. Sometimes syphilis may be diagnosed in its latent period only by means of serology tests.

The Wassermann test is of the greatest importance, its reliability and sensitivity making it the basic test for syphilis. However, this test sometimes also produces nonspecific positive results in persons not affected with syphilis.

Of course, no such errors must be allowed. A wrong diagnosis of syphilis inflicts a severe psychic trauma on the patient. Still greater harm may be done by antisyphilitic treatment in the absence of syphilis. That is why not only Wassermann test, but a complex of 3 serology tests—Wassermann and two flocculation tests—is used in the Soviet Union. These tests are called flocculation tests because the antibody reacts directly with the antigen to produce flocculi. The flocculation tests most commonly used are the Kahn test and the Sachs-Witebsky test. The complex of 3 tests considerably reduces the possibilities of accidental positive results. In most cases the results of Wassermann and the flocculation tests coincide.

Wassermann test and the flocculation tests are based on the deep physico-chemical changes caused by the syphilitic infection in the blood serum.

Since the physico-chemical changes in the serum may be pronounced to various extents the plus sign (+) is used to evaluate the results of the serology tests. The $++++$ (4+) result corresponds to a sharply positive reaction, $+++$ (3+)—positive, $++$ (2+) and (1+)—weakly positive, and $\pm$—dubious. A negative result of a serology test indicates that no physico-chemical changes observed in syphilis have been found. in the tested serum. This does not mean, however, that the particular person has no syphilis. During the latent period of syphilis the changes caused by this disease in the organism, particularly in the patient's blood serum, may be normalised.

During a new attack of the disease the serology tests may become positive again.

Hence, a negative result of a serology test does not always prove that the patient has no syphilis.

Nor are positive results of Wassermann and flocculation tests always signs of syphilis. In some diseases the physico-chemical changes in the blood closely resemble those in syphilis. It is but natural that Wassermann and flocculation tests may prove positive in these diseases. Positive serology tests in the absence of syphilis are called *nonspecific tests.*

Nonspecific positive serology tests are observed in patients with lepromatous leprosy, systemic lupus erythematosus, scarlet fever, relapsing fever, malaria, measles, influenza and malignant tumours in the disintegration stage.

A number of precautions must be taken to avoid an erroneous diagnosis of syphilis in a healthy person, as a result of positive nonspecific tests. For example, in a case of positive serology tests in a person who shows no signs of syphilis and who never knew he had the disease, it is necessary to repeat the tests 2-3 times at intervals of 1 or 2 weeks. At the same time it is necessary to examine the persons who were in sexual or other close contact with the patient. It should also be remembered that nonspecific tests are usually weakly-positive, unstable and, when repeated, may become negative.

Lastly specific tests may be distinguished from nonspecific. The Wassermann test is performed with serum diluted with physiologic solution (1 : 10, 1 : 20, 1 : 40, 1 : 80, etc.). In most cases nonspecific tests produce positive results only in weak solutions. If these precautions are taken, the serology tests are an exceptionally valuable method of diagnosing syphilis.

It is best to take blood for a test on an empty stomach. On the day the blood is taken and the day preceding it the patient must not partake of alcoholic beverages.

Serology tests are extremely useful in mass examinations aimed at discovering syphilitic patients, for example, in cases of venereological expeditions, examinations of pregnant women and of people hired for work in children's institutions and foodpacking plants. It is thus possible to discover and institute treatment of syphilitic patients who not infrequently are unaware of their disease (unknown syphilis).

Serology tests are particularly important in cases of preventive examinations of future donors because among them there may be persons who have latent syphilis and know nothing about it. In many cases serology tests make it possible to discover such patients and prevent them from joining the ranks of donors.

Wassermann and flocculation tests are nearly always positive in patients with early secondary and relapsing secondary syphilis.

In patients with primary syphilis the serology tests change from negative to positive 5-7 weeks after inoculation.

Negative serology tests in the presence of active manifestations of secondary syphilis indicate low resistance of the organism and an unfavourable course of the disease.

In active tertiary syphilis positive serology tests are observed in 60-80 per cent of the patients. In patients with latent secondary and latent tertiary syphilis negative results of serology tests are much more frequent.

Positive serology tests in patients with latent syphilis indicate that the syphilitic infection is active and that the appearance of new active manifestations of the disease is possible.

Serology tests are also very important for evaluating the condition of the syphilitic patient and for judging the results of the administered antisyphilitic treatment.

Regular serology tests of syphilitic patients during treatment (before and after each course of treatment) make it possible to observe the effects produced by the antisyphilitic treatment. After a certain period of antisyphilitic treatment positive reactions usually change to negative reactions. Stable positive serology tests in adequately and regularly treated patients, as a rule, indicate trouble. Such patients need additional examinations and special methods of treatment.

Soviet serologists have elaborated a method of producing and transporting a desiccated serum ("dry drop"), which now makes it possible to perform serology tests in communities where there are no serologic laboratories. Since the territory of the Soviet Union is so vast this method is particularly important and is used mainly in rural hospitals and at physician's assistant's and midwife's stations (see Supplement, p. 337).

A new serology test—*Treponema pallidum* immobilisation test (TPI)—proposed by Nelson and Meyer is very valuable for diagnosing syphilis. It is based on the fact that the antibodies forming

in the organism of the syphilitic patient include *immobilisins*, i.e., antibodies which by acting on the *Treponema pallidum* immobilise it; this can easily be observed under the microscope in dark-field illumination.

The chief merit of the TPI is the infrequency of nonspecific positive results. The TPI is therefore particularly important in cases where the diagnosis of syphilis is established only on the basis of positive results of serology tests without any indications of syphilis in the patient's anamnesis or any clinical manifestations of the disease. Medical workers come across such facts in discovering patients with "unknown syphilis", in serologic examinations of pregnant women (wassermannisation), donors, etc. In such cases positive TPI results confirm the specific character of the positive results of Wassermann and flocculation tests and warrant the diagnosis of syphilis. On the other hand, negative TPI results serve in such cases as grounds for regarding the positive results of Wassermann and flocculation tests as nonspecific.

The shortcoming of the TPI is the complex method of its production, owing to which it can be performed only in large, well-equipped serologic laboratories by specially trained personnel.

That is why serologists the world over are searching for new and simpler serology tests. Some success in this respect has been achieved by the Soviet serologist N. Ovchinnikov, the Polish serologist Metzger and others.

In the Soviet Union the TPI is performed with desiccated serum; this makes it possible to use this valuable method of diagnosing syphilis in communities which do not have any large serologic laboratories.

TREATMENT OF SYPHILIS

The treatment of syphilitic patients is aimed at:

(1) ridding the patients, as quickly as possible, of the communicable manifestations of syphilis and rendering them safe for those around them;

(2) eliminating, as quickly as possible, the active manifestations of syphilis and restoring the functions of the organism impaired by the disease;

(3) preventing phenomena of relapsing and tertiary syphilis and late syphilitic affections of the nervous system;

(4) preventing the birth of children infected with syphilis.

The achievements of modern medicine have made syphilis an entirely curable disease. Today medicine has potent antisyphilitic preparations, including those of penicillin, arsenic, bismuth, mercury and iodine. The effects of these preparations on the disease are so regular that they have been given the designation of *specific* preparations. The treatment of syphilitic patients is also frequently referred to as *specific*.

Fundamental Principles of Treating Syphilitic Patients

1. The treatment of syphilis must begin as early as possible. The sooner the treatment has begun after inoculation, the easier and sooner will the patient be cured. Contrariwise, the later the treatment has begun, the longer the process and the more frequent the failures.

The best results are produced by treatment instituted in the primary, seronegative stage of the disease when the patient has a hard chancre and concurrent bubo, and the serology tests are still negative.

2. Syphilitic patients must be treated for a long time. Disappearance of the external manifestations of syphilis do not as yet indicate a cure.

During the latent period of syphilis the *Treponema pallidum* may long remain in its depot, then become active again and cause affections of the skin, mucous membranes, bones, internal organs and nervous system. However, antisyphilitic treatment must be intermittent. Continuous treatment for a too long time weakens the patient's organism and causes phenomena of intoxication and various complications. Some antisyphilitic preparations possess *cumulative* action, i.e., an ability to accumulate in the organism and cause intoxication when administered for a long time.

Syphilis is therefore treated by separate *courses* with intervals in between. During the intervals the organism rests from the treatment and the antisyphilitic preparations accumulated in the organism are gradually eliminated.

This method of treatment is called *chronically intermittent* or *course* treatment.

Extensive experience has shown that chronically intermittent or course treatment is the best of all the existing methods of treating syphilis.

3. For the treatment of syphilitic patients it is desirable to use not one, but several antisyphilitic preparations. Treatment with one preparation, for example, only penicillin or only bismuth, fails more frequently than does treatment with a combination of several preparations. The reason for it is that the *Treponema pallidum* has the property of adapting itself to new conditions in the organism connected with the treatment and becomes habituated to the preparation. Under these conditions the continued administration of the preparation is no longer effective.

Administration of several antisyphilitic preparations creates the most unfavourable conditions for the *Treponema pallidum* in the organism. That is why the antisyphilitic preparations are alternated for the repeated courses of treatment. For example, if the first course of treatment was administered with bioquinol (a quinine bismuth preparation), the second course of treatment

is administered with bismoverol (another bismuth prepara-tion).

4. Success in the treatment of syphilis can be achieved only by administration of proper doses of antisyphilitic preparations. Administration of excessive doses or reduction of the intervals between the separate doses aggravates the patient's general con-dition and produces various complications. It must not be forgot-ten that increasing the resistance of the organism and strengthen-ing it in its struggle with the *Treponema pallidum* is no less im-portant than acting on the *Treponema pallidum* itself.

The patient is likewise greatly harmed by too small doses of antisyphilitic preparations or by increased intervals between the separate doses. Under such conditions the *Treponema pallidum* may become resistant to these preparations. This always threatens failure of the treatment, i.e., relapses of the disease and develop-ment of late affections of the nervous system and internal organs. The doses administered to the patient must depress the *Treponema pallidum* and at the same time inflict no harm on the patient's or-ganism.

In view of the aforesaid, instructions for the treatment of syph-ilis, establishing doses of antisyphilitic preparations, have been elaborated in the U.S.S.R. However, in prescribing any antisyph-ilitic preparation the individual characteristics of each patient must be taken into consideration.

Penicillin

Today penicillin eliminates the *Treponema pallidum* from the surface of the primary lesion and the syphilids of the second-ary stage faster than does any other antisyphilitic preparation. The active manifestations of syphilis of all stages disappear more rapidly when treated with penicillin than with any other preparation.

The effect of penicillin during the latent period of syphilis is weaker because penicillin is the most active against microbes at the time of their multiplication. In the therapy of syphilis peni-cillin therefore produces the greatest effect in the treatment of patients with active manifestations of the disease, at the time of intense multiplication of the *Treponema pallidum*.

One of the merits of penicillin is that it is well tolerated by patients. With the doses of penicillin used in the treatment of syphilis side effects (headaches, skin eruptions in the form of urti-caria, erythema, etc.) are observed comparatively rarely.

The treatment of patients with active manifestations of syph-ilis is very often accompanied by an "aggravation reaction" — clearer and more plentiful eruptions, elevated temperature, head-ache and weakness. It is particularly clearly marked in patients

with primary seropositive, recent and relapsing secondary, and early congenital syphilis.

To avoid an excessively sharp aggravation reaction, the injections of penicillin are given in half their dose in the first days of treatment, and in one-fourth their dose or even less in cases of congenital syphilis. The aggravation reaction may be dangerous to the patient's health and even life when penicillin is used in the treatment of patients with syphilis of the central nervous system, syphilitic aortitis and tertiary affections of the internal organs, larynx, etc. The patients with such manifestations of syphilis have to be "prepared" for penicillin treatment by preliminary administration of potassium iodide and bioquinol.

Since penicillin is well tolerated it may be administered to syphilitic patients for whom treatment with other antisyphilitic preparations is contraindicated.

In the treatment of syphilitic patients in hospitals penicillin is administered intramuscularly in a dose of 50,000 u every 3 hours.

Ecmonovocillin (a penicillin preparation) is used in ambulant and often in hospital treatment. It is administered intramuscularly in a dose of 600,000 u once a day or 300,000 u twice a day. From 6,000,000 u to 8,400,000 u and even more is administered during a course of treatment, depending on the stage of syphilis and the patient's weight.

Bicillin-1 and bicillin-3 are improved penicillin preparations; after their administration the blood contains adequate antisyphilitic amounts of penicillin for several days.

Bicillin-1 is administered intramuscularly in doses of 1,200,000 u once in 6 days or 2,400,000 u once in 10 days. Bicillin-3 is also administered intramuscularly in doses of 1,200,000 u once in 5 days or 2,400,000 u once in 8 days. The single dose of bicillin (1,200,000 or 2,400,000 u) is divided in half and is injected into both buttocks.

The course doses of penicillin preparations—ecmonovocillin, bicillin-1 and bicillin-3—are the same as those of water-soluble penicillin. The same caution must be exercised in administering these preparations as in administering insoluble suspensions (p. 273).

The course dose of penicillin for patients with seronegative syphilis I is determined on the basis of 100,000 u per 1 kg of the patient's weight, but not less than 6,000,000 u per course of treatment.

For patients with seropositive syphilis I, recent syphilis II and latent syphilis II penicillin is prescribed on the basis of 120,000 u per 1 kg of the patient's weight, but not less than 7,200,000 u per course. For patients with relapsing syphilis II, active syphilis III, latent syphilis III, latent syphilis, neurosyphilis and visceral

syphilis penicillin is prescribed on the basis of 140,000 u per 1 kg of the patient's weight, but not less than 8,400,000 u per course.

Syphilitic children are treated with penicillin by a special method (see Supplement, p. 338).

The potent antisyphilitic action and good tolerance of penicillin have prompted some physicians to treat syphilitic patients with penicillin alone and neglect all other antisyphilitic agents. Experience has demonstrated the fallacy of this method since some patients treated with penicillin alone had relapses of syphilis and developed affections of the nervous system. The American syphilologist Charles Dennie admits that the hullaballo raised around penicillin in the U.S.A. by businessmen and private practitioners led to an ungrounded relinquishment of preparations of arsenic, bismuth, mercury and iodine. Dennie also emphasises that penicillin alone cures only 80 per cent of the syphilitics in the U.S.A., while the remaining 20 per cent are actually left untreated.

In the Soviet Union penicillin is the principal agent in the treatment of syphilis, but other antisyphilitic agents are also used. Penicillin combined with other antisyphilitic preparations produces the best results.

Arsenicals

For several decades, since 1909, complex organic arsenicals so-called *salvarsan* (*arsphenamine*) and *osarsol* (*acetarsol*), have been the principal agents in the treatment of syphilis. These agents produced vigorous therapeutic effects, but have also caused a number of side effects and complications often impossible to foresee and prevent. For this reason, with the appearance of penicillin, which is much more effective than arsphenamine preparations and much less frequently productive of complications, these preparations began to recede into the background.

Today arsphenamine preparations are very rarely used in the treatment of syphilitic patients and are administered mainly in cases of individual indications—intolerance of penicillin and other antibiotics by the patients, contraindications for antibiotics, etc. The arsphenamine preparations produced in the Soviet Union are myarsenol (sulfarsphenamine) and novarsenol (neoarsphenamine).

Novarsenol is dispensed in glass ampules of 0.15, 0.3, 0.45 and 0.6 g. The preparation is a loose orange-yellow powder freely soluble in water. In the presence of air novarsenol oxidises and becomes very toxic. The contents of an ampule are dissolved in 5 ml of distilled or freshly sterilised water regardless of the dose and are administered intravenously over a period of 1.5-2 minutes. The preparation must be injected very carefully so that it may not accidentally get into the subcutaneous tissue because even a small amount of the solution getting under the skin causes intense pain, swelling, infiltration and sometimes formation of an ulcer.

The dose of novarsenol is calculated at 0.1-0.12 g per day and is administered mainly to patients with active manifestations of primary or secondary syphilis, and only to vigorous, healthy men who can tolerate the treatment. The first injection given to women is usually 0.15 g, and that given to men is 0.3 g; at each injection the dose is increased 0.15 g. The highest single dose is 0.45 g for women and 0.6 g for men.

The intervals between the single doses are calculated on the basis of the daily doses. A course of treatment consists in administration of 3.5-5.5 g of the preparation.

The higher doses of 5-5.5 g for men and 4.5-5 g for women are prescribed to patients with active manifestations of primary and secondary syphilis provided they are in good general condition and can tolerate the treatment. In cases of any disturbances in the patient's general condition the course dose is somewhat reduced. Patients with tertiary syphilis are prescribed 3.5-4 g of novarsenol per course of treatment.

Myarsenol is chemically closely related to novarsenol. It is a straw-yellow, loose powder, freely soluble in water, and is dispensed in ampules of 0.15, 0.3, 0.45 and 0.6 g.

Myarsenol is dissolved regardless of the dose in 2 ml of distilled, freshly sterilised water and is administered intramuscularly. The daily, single and course doses of myarsenol are the same as those of novarsenol.

Arsphenamine preparations are very potent and are not always well tolerated by the patients. Before instituting treatment with novarsenol or myarsenol it is necessary to make sure that these preparations are not contraindicated for the patient.

There are *absolute* contraindications, i.e., when these preparations cannot be used at all, and *relative* contraindications, i.e., when the preparations may be used with caution (see Supplement. p. 338).

Osarsol is an organic arsenical closely related to arsphenamine preparations but differing from them in chemical structure.

Osarsol is inferior to novarsenol and myarsenol in its effects on syphilis. It is a white powder, insoluble in water and is given per os in a dose of 1 g a day—0.5 g in the morning and 0.5 g in the evening, 1 hour before meals. The preparation is administered 5 days in succession and is followed by a 3-day interval. A course of treatment consists of 8 such 5-day cycles with 3-day intervals (40 g of the preparation).

In the first cycle administration of osarsol begins with small doses: 0.25 g in the morning of the first day, 0.25 g in the morning and 0.25 g in the evening of the second day, 0.5 g in the morning and 0.25 g in the evening of the third day, 0.5 g in the morning and 0.5 g in the evening of the fourth and fifth days.

Children are prescribed arsphenamine preparations in doses reduced according to age (see Supplement, p. 339).

Arsenicals may produce various complications in syphilitic patients. The complications may arise: (1) directly during administration of the preparations or a few hours after their administration, (2) several days after administration of the preparations, and (3) several weeks after the end of the course of treatment.

The following are the most common complications:

1. *Nitritoid crisis* which usually develops during injection or immediately following and is marked by redness and edema of the patient's face and neck, rapid pulse and dyspnea. In some cases there are pain and constriction in the chest, a fear of death, urticarial eruption and vomiting. All these phenomena disappear within 10-15 minutes. In cases of a nitritoid crisis the patient must quickly be given a subcutaneous injection of 0.5-1 ml of an adrenalin solution (1 : 1,000) and 1 ml of a 10 per cent caffeine solution.

2. *Fever* which may arise several hours after the first administration of novarsenol or myarsenol is most commonly observed in patients with recent or relapsing secondary and with primary seropositive syphilis. This fever is due to mass disintegration of the *Treponema pallidum* after the first administration of an arsenical and is called an *aggravation reaction*. Simultaneously with the fever the syphilitic eruptions on the skin usually become more numerous and more clearly defined.

The fever may also develop between the 7th and 12th days after the first injection—so-called *fever of ninth day*. It is often accompanied by various eruptions on the skin. These eruptions are called *erythema of ninth day*.

The fever and erythema of ninth day are based on the patient's sensitivity to arsphenamine preparations and are treated with desensitising agents—dimedrol, sodium hyposulfite, calcium chloride and ascorbic acid.

In some patients the temperature rises after every administration of novarsenol and myarsenol. This attests poor tolerance of arsphenamine preparations and is called *arsphenamine fever*.

3. *Headache.* After administration of arsphenamine preparations patients sometimes develop headache without a rise in temperature. Some patients have headache after every injection, which denotes poor tolerance. Such patients are prescribed intravenous injections of glucose, sodium hyposulfite, and vitamins C and B_1. Administration of arsphenamine preparations must be discontinued.

4. *Arsphenamine hemorrhagic encephalitis* is the severest complication of treatment with arsenicals.

Hemorrhagic encephalitis begins (usually after 2-4 injections) with headache on injection day or 2-3 days after the injection.
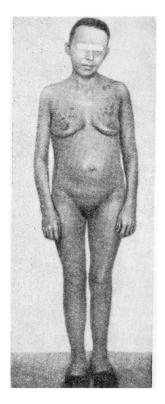
Fig. 150. Erythroderma
caused by treatment with
arsenicals

The headache rapidly increases in intensity and becomes unbearable. The patient loses consciousness, develops convulsions, vomits, and performs involuntary movements. In many cases death ensues between the 3rd and 5th days.

Hemorrhagic encephalitis is treated by bleeding (300 ml), lumbar puncture with removal of 20-40 ml of fluid, administration of adrenalin, glucose, caffeine, and induction of narcotic sleep by administration of hexenal (hexobarbital) and medinal (barbital sodium) (M. Rozentul and others).

5. *Arsenical jaundice* may occur in she course of treatment or after the end of treatment, most commonly several weeks after the end of the course, and may last from 2 weeks to 3 months. In most of these cases it is an infectious disease—Botkin's disease (infectious hepatitis). It is often inoculated as a result of poor sterilisation of the syringes and needles used in injections. Arsphenamine plays the part of a predisposing factor by exerting a toxic influence on the organism, the liver in particular. Prevention of arsenical jaundice consists in thorough sterilisation (by boiling for 30 minutes) of the syringe, needles and vessels used in dissolving novarsenol and myarsenol, prescription of a diet rich in carbohydrates and vitamins, and in daily injections of glucose and ascorbic acid.

6. *Arsenical dermatitides* may be early, if they appear several hours or days after injection, or late, if they develop several weeks after the treatment.

Mild, moderate and severe dermatitides are distinguished. In mild and moderate dermatitides the eruptions consist of macules, blisters and follicular papules; itching, sometimes quite intense, is observed. Mild dermatitides disappear within a few days; moderate dermatitides last up to 2 weeks. On the appearance of dermatitis treatment with arsphenamine preparations must be discontinued.

Severe arsenical dermatitis—arsenical erythroderma—develops at the end of the course of treatment or some time after the end of treatment.

The patient's entire skin becomes bright-red and edematous, vesicles and exudation appear, intense itching, burning and fever develop (Fig. 150). The condition is very often accompanied by secondary pyoderma—impetigo, folliculitides, furuncles and, sometimes, sepsis. The disease lasts from a few weeks to several months.

Proper treatment excludes the possibility of death and considerably reduces the duration of the disease. The patients are given blood transfusions (100-200 ml once in 6-7 days) and are administered penicillin (a total of 4,000,000-6,000,000 u), ascorbic acid, sodium hyposulfite, dimedrol, adrenalin or ephedrine, campolon (aqueous liver extract) and vitamins B_1, B_2 and PP. Lotions and "zinc oil" are applied locally.

Arsenical dermatitides are a manifestation of hypersensitivity of the patient's organism to arsenicals.

Of the arsenicals used in the treatment of syphilis novarsenol causes complications the most frequently, myarsenol—less frequently, and osarsol the least frequently.

To avoid complications, the patient must be prescribed a proper regimen—enough rest, regular hours outdoors, at least 7 or 8 hours of sleep a day, and regular meals with a lot of carbohydrates, fresh vegetables and fruits, and vitamins.

Bismuth Preparations

Bismuth preparations are active antisyphilitics; they produce good therapeutic effects in all stages of syphilis and in effectiveness are second to arsenicals.

They are well tolerated by patients and produce complications much less frequently than do arsenicals.

Bioquinol, bismoverol (monobismuth salt in neutral vegetable oil) and pentabismol are the bismuth preparations most commonly used in the Soviet Union.

Bioquinol is quinine bismuth iodide. It is a bright-red powder insoluble in water; it is dispensed as an 8 per cent suspension in sterile oil. Before administration the vial of bioquinol is placed in hot water (40-45°C) and is then thoroughly shaken to produce a uniform suspension.

Bioquinol is administered intramuscularly. To avoid unpleasant and dangerous complications due to penetration of the oil suspension into an artery or vein, it is necessary to take the following precautions. Injections are made into the exterosuperior part of the buttock (Fig. 151) with a long needle (5-6 cm). The preparations must not be administered into any other parts of the buttock to avoid injuring the blood vessels and nerve trunks running through those parts. The needle is introduced into the muscle and firmly fixed with the fingers of the left hand, after which the syringe

is removed from the needle. If no blood shows from the needle for 1-2 minutes, the syringe is reset on the needle, the latter continuing to be firmly fixed in its immobile position, and bioquinol is injected into the muscle. But, if blood shows from the needle,
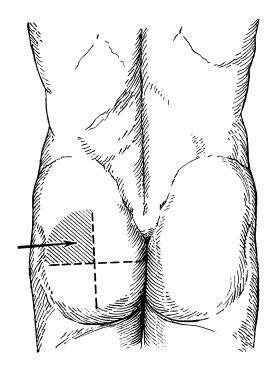
Fig. 151. Part of the buttock for intramuscular injections

the needle is removed and the injection is made at another site.

Bioquinol is administered according to all rules of asepsis and antisepsis, i.e., the site of the injection is first disinfected with cotton moistened in alcohol, and the syringe and needle are sterilised by boiling. The preparation is prescribed in a dose of 1 ml per day, but is administered in a dose of 3 ml once in 3 days. A dose of 2 ml is recommended for the first injection. Pregnant women are usually administered 2 ml of the preparation every other day. The amount of bioquinol for the course of treatment is 40-50 ml.

Patients who live in the countryside and have to walk several kilometres for the treatment may be given single injections of

5 ml provided they are in good general condition and can tolerate the treatment. In such cases this dose is divided in half and 2.5 ml of bioquinol is injected into each buttock. It is generally inadvisable to inject more than 3 ml into a buttock at one time because with larger amounts of the suspension bioquinol is poorly absorbed and may injure the muscles and nerve trunks.

Bioquinol is very well tolerated by most patients; the injections of this preparation are almost painless.

Bismoverol, a yellowish-white powder, insoluble in water, is a monobismuth tartrate. It is dispensed in the form of a 7.5 per cent suspension in sterile oil and is administered intramuscularly. The dose of bismoverol is 1 ml every other day in the first half of the course and 1 ml once every third day in the second half of the course; 16-20 ml of the preparation is administered for a course. It is usually well tolerated by patients.

Pentabismol is a new bismuth preparation; unlike bioquinol and bismoverol, it is a water-soluble bismuth compound. It is administered intramuscularly in a dose of 2 ml every other day, 40-50 ml per course of treatment.

Bismuth preparations are contraindicated in cases of severe general diseases, acute infections, renal diseases, diseases of the oral mucosa and clearly marked alveolar pyorrhea.

Mercurials

Mercurials are much weaker antisyphilitics than are bismuth preparations and, moreover, quite frequently cause complications, for which reason they have only recently begun to be used in rare cases to treat syphilitic patients.

Mercurials may be used as soluble and insoluble compounds for intramuscular injections and as mercurial ointment for inunction.

Soluble mercurials. Injections of soluble mercurials are relatively painless. The most commonly used soluble mercurials are mercuric cyanide, a 2 per cent solution of which is administered intramuscularly in a dose of 1 ml every other day, a course of treatment consisting of 20 injections (20 ml of the solution), mercuric oxycyanide, which is administered in 1 and 2 per cent solutions, 1 and 2 per cent solutions of mercury bichloride and mercuric iodide.

Insoluble mercurials. The negative feature of these preparations is that their administration is painful, complications develop more frequently and they are sometimes graver.

The most commonly used insoluble mercurials is mercuric salicylate which is administered as a 10 per cent solution in sterile peach or almond oil. Glass beads are put into the vials with mercuric salicylate suspension to facilitate its shaking.

The dose of the suspension for the first injection is 0.5 ml; the second injection is made 2-3 days later with 1 ml of the suspension. The subsequent injections consist of 1 ml of the suspension once in 5-7 days, 10-15 ml of the preparation for a course of treatment.

Injections of soluble and insoluble mercurials have largely lost their significance and are now very rarely prescribed to syphilitic patients. Mercurial inunctions are used more frequently.

Mercurial inunctions. A mercurial ointment containing 33 per cent metallic mercury on a fatty base is used for mercurial inunctions. The dose for each inunction is 4-5 g of the ointment which is rubbed into different parts of the skin in a certain succession 4-5 days running. On the first day the ointment may be rubbed into the flexor surface of the forearms, on the second day—into the shoulders, on the third day—into the lateral surfaces of the chest, on the fourth day—into the abdomen, and on the fifth day—into the medial surface of the thighs. In choosing the sites for inunction of the mercurial ointment it is necessary to avoid the parts of the skin covered with a lot of downy hair. The portion of ointment is always divided into two equal doses, one to be rubbed in on the left side and the other on the right side. On the sixth day the inunctions are interrupted, the patient must take a bath, and the inunctions are then resumed. A course of treatment consists of 30-40 inunctions.

The method of inunction is indicated in the treatment of patients with neurosyphilis, visceral syphilis, and stable positive serology tests.

The mercurial ointment must be rubbed in the direction of the growth of downy hair, slowly (over a period of 30-40 minutes), vigorously and till it dries. This method produces good results only with a proper inunction technique. The greatest shortcoming of this method is that the treatment is administered by the patient himself, for which reason it is difficult to control.

The contraindications for mercurials are essentially the same as those for bismuth preparations. Inunctions of mercurial ointment are also contraindicated by skin diseases and abundant hairiness.

Complications Produced by Bismuth Preparations and Mercurials

1. *Complications at the site of administration of the preparation.* In some patients the preparations of bismuth and, especially, mercury cause formation of painful infiltrates in the gluteal muscles. Still more painful are the infiltrates in the subscutaneous tissue, which are formed when the injections are made with insufficiently long needles. Formation of infiltrate around the administered preparation is undesirable because the infiltrate slows the absorp-

tion of the preparation into the blood and thereby reduces the effect of the treatment. Infiltrates not infrequently leave cicatricial changes in muscle tissue making absorption of the preparation during the subsequent injections more difficult.

Mercurials, especially insoluble preparations, cause formation of infiltrates much more frequently than do bismuth preparations. Diathermy, irradiation with a sun lamp and heat are used for the treatment of the infiltrates.

Abscesses are sometimes formed at the sites of injections. The cause of this complication lies mainly in violation of the rules of asepsis during the injections.

Use of improper techniques of injecting bioquinol, bismoverol and mercuric salicylate suspension may give rise to serious complications as the result of penetration of the oil suspension into the lumen of a blood vessel, which leads to obstruction of the terminal branch of the artery with oil and necrosis of the tissues in this area. The injection is very painful; soon after the injection the buttock develops edema, the skin becomes hot, tense, and covered with purple macules and with lines forming a net. These phenomena are followed by deep necrosis of the tissues with very slow healing.

2. Penetration of the oil suspension into the lumen of a vein gives rise to obstruction of one of the branches of the pulmonary artery. During the injection or soon afterwards the patient develops a sudden attack of coughing and pain in the chest or side, sometimes dizziness and weakness. This may subsequently be followed by pneumonia or a pulmonary infarct.

3. *Gingivitides and stomatitides*. Mercurials very often cause complications in the oral mucosa. Bismuth preparations cause them much less frequently. The disease usually begins with affection of the gums which grow loose and red, their papillae become swollen and acquire a purplish hue. These phenomena are subsequently followed by pain and bleeding under pressure and during eating. Pressure on the gingival papillae results in a discharge of drops of pus. The redness extends to the other parts of the mucosa. Very painful erosions which may subsequently develop into ulcers appear on the mucosa of the cheeks, most commonly at the sites of junction of the molars. The patient has an offensive breath. The sharp pain makes speaking and eating difficult.

Gingivitides and stomatitides most commonly develop in patients who before the beginning of the treatment had alveolar pyorrhea, carious teeth, diseases of the oral mucosa, and in cases of inadequate oral hygiene.

These complications are prevented by sanitation of the oral cavity before the beginning of antisyphilitic treatment and strict observance of oral hygiene. The mouth must be rinsed after each meal and brushed with tooth powder for the night.

Gingivitis and stomatitis are treated by disinfecting gargling and warm irrigations of the oral cavity with a 2 per cent soda solution twice a day.

The soda solution is poured into an Esmarch can which is placed at a height of 1-2, and all parts of the oral mucosa are thoroughly irrigated with the solution through a glass nozzle. Painting the affected parts of the mucosa with a novarsenol solution (0.6 g per 5 ml of water) several times per day often helps very much in cases of erosions, ulcers and putrefactive phenomena. Administration of mercurials or bismuth preparations is suspended until disappearance of the phenomena of gingivitis and stomatitis. Vitamins C, PP and B_2 are administered per os.

Bismuth preparations very often cause the appearance of a "bismuth border"—a greyish strip along the edges of the gums. In the absence of bismuth complications the appearance of the bismuth border is no hindrance to continuation of the treatment, but it requires caution and careful watching of the patient.

4. *Nephropathies*—renal complications—are more frequently observed during the treatment of syphilitic patients with bismuth preparations. In most cases bismuth nephropathies run a mild course and disappear relatively soon after cessation of the treatment with bismuth. "Bismuth cells" in the precipitate of the urine—degenerated cells of renal epithelium—serve as the earliest sign of nephropathy, albumin and casts appearing in the urine much later. Mercurial nephropathies occur less frequently. Their first signs are an increased urinary output and albumin in the urine.

To prevent severer nephropathies, the treatment must be suspended for several days upon the first signs of renal irritation. A 3-4 per cent potassium iodide solution in tablespoonful doses is administered for better elimination of bismuth and mercury from the organism.

The urine must be analysed once a week all through the course of treatment in order that developing nephropathies may be noticed in due time.

Treatment with mercurials is also accompanied by weakness, anemia, poor appetite, loss of weight, abdominal pain, diarrhea and dermatitides. Mercurial inunctions often result in folliculitides and furunculosis.

Iodine Preparations

Iodine preparations are much inferior to bismuth preparations and mercurials in their effects in primary and secondary syphilis.

In the treatment of syphilitic patients iodine preparations play an important part just the same because of the effects they produce in tertiary syphilis, affections of the nervous system and the internal organs. Iodine preparations also favour resorption

Fig. 152. Acne caused by treatment with iodides

of enlarged lymph nodes in patients with primary and secondary syphilis, and of the infiltrate persisting at the site of the primary lesion.

Iodine preparations likewise produce favourable effects in headaches, ostalgia, periostitides and fever in patients with secondary syphilis. They are also very helpful in the treatment of patients with latent syphilis. They are usually administered during intervals between courses of treatment with bismuth, penicillin and other preparations because they favour better elimination of these preparations from the patient's organism.

Potassium iodide and sodium iodide are the *iodide alkalis* which produce the best effects in the treatment of syphilitic patients. They are administered per os, usually as a course of treatment in a dose of 1 tablespoonful of a 2-3 per cent solution in half a glassful of milk 3 times a day after meals for 3-4 weeks. The dose is then gradually increased, the patient being given

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stronger solutions of iodide alkalis (up to 7-8 per cent, 1 tablespoon-ful 3 times a day). Sodium iodide may also be administered intravenously in a daily dose of 5-10 ml of a 10 per cent solution.

In some cases iodide alkalis cause complications, such as *iodism* —coryza, conjunctivitis, excessive lacrimation and "iodide acne". The iodide acne consists of papules and pustules resembling acne vulgaris and distributed over the face, chest and back (Fig. 152). In cases of iodism administration of iodine preparations is temporarily suspended and calcium chloride and ascorbic acid are administered. Ascorbic acid must be prescribed prophylactically from the very beginning of iodine treatment.

If potassium iodide and sodium iodide are not tolerated by the patients, they may be replaced by peroral administration of a 10 per cent iodine tincture (5-50 drops 3 times a day after meals) or sajodin (0.5-1 g 3 times a day). These iodine preparations are tolerated by patients better than potassium iodide and sodium iodide, but they are much less effective.

Methods of Treating Syphilitic Patients

In the Soviet Union syphilitic patients are treated by the chronically intermittent method.

The treatment of patients with active manifestations of primary and secondary syphilis usually begins with penicillin therapy. The treatment of patients with tertiary syphilis, visceral syphilis and late neurosyphilis begins with administration of iodine or bismuth preparations, which is followed by penicillin therapy.

The Instructions of the U.S.S.R. Ministry of Public Health on the treatment of syphilitic patients envisages 4 basic variants of treatment.

1. *Treatment of syphilitic patients with penicillin and bismuth preparations.* The combined courses of treatment begin with injections of water-soluble penicillin or ecmonovocillin and are followed by injections of one of the bismuth preparations (see Fig. 153). The number of combined courses of treatment depends on the stage of syphilis (see Supplement, p. 340).

2. *Treatment of syphilitic patients by repeated courses of penicillin therapy.* Syphilitic patients may be treated with penicillin without bismuth preparations. This is particularly necessary in cases where administration of bismuth preparations is undesirable because of the patient's affection with tuberculosis, diseases of the liver or kidneys, or other contraindications, when the patient is past 50 years of age or does not tolerate bismuth preparations. In such cases the treatment consists in repeated courses of water-soluble penicillin or ecmonovocillin therapy (see Fig. 154).

DIAGRAM SHOWING TREATMENT OF SYPHILITIC PATIENTS WITH PENICILLIN AND BISMUTH PREPARATIONS

Seronegative syphilis I
Seropositive syphilis I
Recent syphilis II
Latent syphilis II
Relapsing syphilis II
Early neurosyphilis
Active syphilis III
Latent syphilis III
Latent syphilis (d'emblée)
Late neurosyphilis
Visceral syphilis
Late congenital syphilis

□□ Course of penicillin or ecmonovocillin injections
□□ Course of bioquinol, bismoverol or pentabismol injections

TREATMENT OF CHILDREN

Congenital syphilis of infants
Congenital syphilis of early childhood
Late congenital syphilis
(5-15 years of age)

Fig. 153. Diagram showing treatment of syphilitic patients with penicillin and bismuth preparations

DIAGRAM SHOWING TREATMENT OF SYPHILITIC PATIENTS WITH PENICILLIN

Seronegative syphilis I
Seropositive syphilis I
Recent syphilis II
Latent syphilis II
Relapsing syphilis II
Early neurosyphilis
Latent syphilis (d'emblée)
Active syphilis III
Latent syphilis III
Late neurosyphilis
Visceral syphilis
Late congenital syphilis

□□ Course of penicillin or ecmonovocillin injections

TREATMENT OF CHILDREN

Congenital syphilis of infants and congenital syphilis of early childhood
Congenital syphilis of early childhood
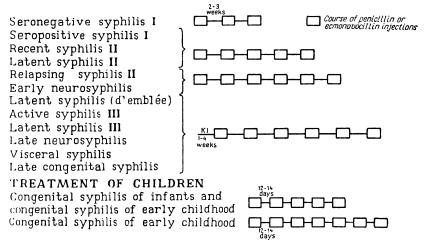
Fig. 154. Diagram showing treatment of syphilitic patients with penicillin

The total number of courses of treatment depends on the stage of syphilis (see Supplement, p. 340).

3. *Treatment of syphilitic patients with bicillin.* Bicillin-1 and biccilin-3 are good antisyphilitics and may be administered even to outpatients. They may be used for repeated courses of treatment without bismuth preparations (see Fig. 155). The number of courses of bicillin treatment also depends on the stage of syphilis (see Supplement, p. 340). Since bicillin possesses greater antisyphilitic activity than do water-soluble penicillin and ecmonovocillin the total number of courses of bicillin treatment is somewhat less than that in cases of treatment with water-soluble penicillin. This can be clearly seen by comparing Fig. 154 and Fig. 155.

4. *Treatment of syphilitic patients with bicillin and bismuth preparations.* Treatment with repeated courses of bicillin is not always sufficiently effective in late forms of syphilis (relapsing syphilis II; active syphilis III; latent syphilis III; latent syphilis; neurosyphilis and visceral syphilis). The patients with these forms of syphilis are therefore often treated by combined courses of bicillin and bismuth preparations (see Fig. 156). The patients are first given injections of bicillin and then of bismuth preparations, a total of 6 combined courses of treatment (see Supplement, p. 340).

In treating syphilitic patients by any of the four above-mentioned methods it is always necessary to take into consideration the patient's general condition, his ability to tolerate the treatment and the speed with which positive serology tests become negative.

If the patient is debilitated or cannot tolerate the treatment, it is best to employ the second or third method of treatment, i.e., administer repeated courses of penicillin or bicillin treatment without resorting to bismuth preparations.

When positive serology tests fail to become negative after the third course of treatment, the treatment must be supplemented with nonspecific treatment (see p. 283). Strong mercurial ointment inunctions may sometimes be successfully used. In cases of relapsing syphilis II, tertiary active and other forms of late syphilis nonspecific therapy should be added to specific treatment from the very outset.

In addition to the four basic methods of treating syphilis treatment with mixed arsphenamine-bismuth courses with or without penicillin may be administered. However, this method of treatment is now very rarely used since most patients do not need it, the treatment by this method takes much longer, the treatment is much less tolerable and complications are observed much more frequently. In the intervals between the courses of treatment with penicillin or bismuth preparations it is well to prescribe iodine preparations, especially for patients with tertiary and other forms of late syphilis.

DIAGRAM SHOWING TREATMENT OF SYPHILITIC PATIENTS WITH BICILLIN

Seronegative syphilis I
Seropositive syphilis I
Recent syphilis II
Latent syphilis II
Relapsing syphilis II
Early neurosyphilis
Active syphilis III
Latent syphilis III
Latent syphilis (d'emblée)
Late neurosyphilis
Visceral syphilis
Late congenital syphilis

2-3 weeks

KI 1-4 weeks

☐ Course of penicillin or ecmonovocillin injections
☑ Course of bicillin-1 or bicillin-3 injections

TREATMENT OF CHILDREN

Congenital syphilis of infants and congenital syphilis of early childhood

12-14 days

Late congenital syphilis
(5-15 years of age)

KI 1-4 weeks

Fig. 155. Diagram showing treatment of syphilitic patients with bicillin

DIAGRAM SHOWING TREATMENT OF SYPHILITIC PATIENTS WITH BICILLIN AND BISMUTH PREPARATIONS

Relapsing syphilis II
Active syphilis III
Latent syphilis III
Latent syphilis (d'emblée)
Late neurosyphilis
Visceral syphilis
Late congenital syphilis

KI 1-3 weeks

1 month

☑ Course of bicillin-1 or bicillin-3 injections
☐ Course of bioquinol, bismoverol or pentabismol injections
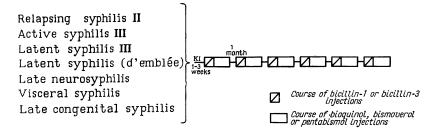
Fig. 156. Diagram showing treatment of syphilitic patients with bicillin and bismuth preparations

Treatment of pregnant women infected with syphilis requires particular attention (see Supplement, p. 342). Penicillin treatment is tolerated well by pregnant women, treatment with bismuth preparations satisfactorily.

Children born without symptoms of congenital syphilis but of inadequately treated mothers are given preventive treatment in the form of 3 penicillin courses and one course of bioquinol injections.

Antisyphilitic treatment is usually well tolerated by children. The doses of all the aforementioned preparations are reduced in accordance with their age (see Supplement, p. 338).

Preventive treatment is administered to persons who had sexual intercourse with syphilitic patients exhibiting contagious symptoms. It consists of one course of penicillin therapy or two courses of mixed therapy for men and three courses for women.

Preventive treatment is expedient only if no more than 15 days have elapsed since the sexual intercourse in which inoculation was possible. If more than 15 days have elapsed since the dangerous contact, the treatment is the same as that of patients with seronegative syphilis I.

Preventive treatment is sometimes administered to persons who have been in close nonvenereal contact with patients in contagious stages of syphilis. For example, if a healthy person used the same plate, cup or spoon as did a syphilitic with contagious phenomena on the lips or in the mouth, the danger of infection is great and preventive treatment is indicated.

In some cases, for example, in the treatment of patients with relapsing syphilis, neurosyphilis or persistently positive serology tests, *nonspecific methods* are used in order to increase the resistance of the organism. For this purpose fever is produced artificially by injections of sulfur, inoculation with malaria or use of high-frequency currents.

Injections of a 2 per cent sulfur (chemically pure) suspension in sterile oil are most commonly used.

The injections are made intramuscularly in doses of 0.3-0.5-0.7-1-1.5-2 ml with intervals of 2-5 days, depending on the reaction to the preceding injection.

Subcutaneous insufflation of oxygen, injections of milk, and exposure to a mercury vapour lamp are also used.

Prognosis and Criterion of Cure in Syphilis

Syphilis may be definitely considered a curable disease.

V. Tarnovsky, originator of Russian syphilology, traced the fate of 50 syphilitic patients over a period of 40 years and found that with adequate treatment these patients long enjoyed good health, died at a very old age and left healthy offspring.

The possibilities of curing syphilis vary with its different stages; a cure can be easily effected in its early forms and is much more difficult in its late forms. It is not always possible completely to arrest the process and restore the functions destroyed by the disease in patients with parenchymatous neurosyphilis and severe tertiary affection of the internal organs and the nervous system.

The possibilities of curing syphilis also depend on the quality of the antisyphilitic treatment. Unfinished and irregular treatment, as well as inappropriate doses of antisyphilitic preparations reduce the patient's chances for recovery, lead to complications in the process of treatment and may make the *Treponema pallidum* resistant to the therapeutic preparations.

An enormous role in the cure of syphilis is played by the patient's general condition, as well as his living and working conditions.

How can a patient's recovery from syphilis be determined?

If the patient has taken all the necessary treatment, exhibits no syphilitic symptoms and the serology tests are negative, the treatment is terminated, but the patient is kept under observation for several years.

Patients with primary seronegative syphilis are kept under observation for at least 2 years, with primary seropositive, recent secondary, relapsing secondary and active tertiary syphilis—at least 3 years, and with congenital syphilis—10 years.

During the observation period the patient must periodically (once in 3 months during the first year and once in 3-6 months during the subsequent years) report for a medical examination and serology tests.

A cerebrospinal fluid test is recommended one year after the end of the treatment.

If no syphilitic symptoms appear during the aforesaid period of observation, the patient is once more thoroughly examined, the examination including roentgenoscopy of the chest and a consultation with a neuropathologist and an ophthalmologist.

If the results of the final examination are favourable, the patient may be taken off the records.

YAWS

Yaws (frambesia, pian) is caused by a special microorganism — *Treponema pertenue*—closely related to the *Treponema pallidum*. This disease is widespread in a number of tropical countries. Infection with yaws is favoured by slovenliness, ignorance of sanitation, poverty and overcrowded living conditions. According to the World Health Organisation (1960), 50 million people had yaws during 1945-1960; in some tropical countries up to 10 per cent of the total population was infected with the disease.

Yaws is not a venereal disease, but we are dealing with it here because it has features similar to those of syphilis. The disease is transmitted through various forms of day-to-day contact of a healthy person with a yaws patient or with an object contaminated by a yaws patient, as a result of which the *Treponema pertenue* gains entrance into the organism through an injury in the skin — abrasion, wound, insect bite, erosion or ulcer. It is believed that the *Treponema pertenue* cannot penetrate into the organism through intact skin. According to some investigators, fleas may be vectors of the infection by biting yaws patients and then healthy people. The disease most commonly affects children and adolescents.

The incubation period of yaws is usually 2-4 weeks. During the incubation period the patient may exhibit prodromal symptoms—indisposition, weakness, headache, elevated temperature, pain in the joints. This period is followed by formation of a reddish papule at the site of entrance of the *Treponema pertenue* into the skin. After this several new papules appear around the first one. The papules enlarge, coalesce and, due to exudation of a seropurulent discharge, become covered with thick yellow crusts. Removal of the crusts reveals soft ulcers with a meat-red granular surface and even edges. These lesions are known as primary yaws papules or "mother yaws". Some patients exhibit a moderate enlargement of regional lymph nodes. The "mother yaws" may be accompanied by pain or itching. Upon removal of the crusts from the primary yaws papules it is not difficult to see the *Treponema pertenue* in the discharge in dark-field illumination. The "mother yaws" usually localise on exposed parts of the skin, especially on the lower part of the shanks and on the face.

Within a few months the primary ulcers heal, the papullar infiltrate is resorbed and only a small scar or cicatricial atrophy

remains at the site of the "mother yaws"; the scar may subsequently be replaced by hyperpigmentation.

From one to three months after the appearance of the primary papule the patients develop eruptions called secondary yaws. Before their appearance the patient may again experience weakness, and have headaches and other health disturbances. The secondary eruptions of yaws are small, reddish, soft papules. They may disappear within a few weeks, leaving macules with branny desquamation, or they may enlarge to the size of a hazelnut and become covered with luxuriant vegetations which make them look like raspberries or cauliflower. The purulent discharge of these vegetations dries into thick, yellowish or brown crusts. The papules often coalesce, forming patches, rings or arches. The eruptions on the palms and soles of the feet often become covered with massive horny layers. Sometimes they become exudative and covered with purulent crusts. The secondary yaws eruptions sometimes cause pain or itching.

In most cases the secondary yaws papules disappear even without treatment within 3-6 months in children, and somewhat more slowly (within close to a year) in adults. The papules leave depigmented macules which later become hyperpigmented. In some patients secondary papules keep recurring for several years.

As a rule, no secondary yaws eruptions appear on mucous membranes.

Several months or even years after the secondary stage of yaws some patients develop the tertiary stage of the disease. In such cases ulcerative nodes greatly resembling syphilitic gummas form in the subcutaneous adipose tissue. Gumma-type lesions in the hard and soft palate, shank bones, nose, etc., are often observed. Necrosis of these lesions leads to extensive destruction of tissues, formation of deep ulcers and disfiguring scars, limitation of mobility of the extremities, etc.

In establishing the diagnosis of yaws it is necessary to distinguish this disease from syphilis with which it has common features —close resemblance of the *Treponema pertenue* to the *Treponema pallidum*, similarity of the clinical manifestations of yaws with some symptoms of secondary and tertiary syphilis, and positive serology tests—Wassermann and flocculation tests. The exclusively nonvenereal character of the infection, the raspberrylike appearance of the secondary papules covered with vegetations and thick yellow crusts, the absence of lesions in the mucous membranes, and of affections of the cardiovascular and nervous systems, and the absence of congenital yaws help to distinguish yaws from syphilis.

Yaws patients are treated mainly with penicillin preparations, usually one injection of about 1,000,000-2,000,000 u sufficing to effect a cure.

The diseases caused by *Treponemas* (*bejel*, *njovera*, *gangosa*) which greatly resemble the *Treponema pertenue* and occur in some tropical countries present a clinical picture which is very similar to that of yaws and are regarded by most investigators as varieties of yaws.

Pinta, a disease most frequently seen in tropical America, differs from yaws more than the aforesaid diseases. It is caused by a special *Treponema* (*Treponema carateum*) which is morphologically identical with the *Treponemas* of syphilis and of yaws. From 7 to 10 days after inoculation a red papule forms at the site of entrance of the *Treponema carateum* into the skin (usually on the shanks or other exposed parts of the skin); the papule enlarges over a period of 2-3 months and develops into an elevated, scaling, red patch around which new macules and papules appear. Several months, a year, or even more, after the inoculation scaling red patches—second stage of pinta—appear mainly on the limbs or on the face. The second stage is subsequently followed by the third stage which is manifested mostly in pigmentation disorders. Bluish and less frequently brownish macules appear and are later replaced by depigmentation. The depigmented macules may persist for a long time. It is believed that pinta is transmitted through bites of fleas and sandflies. Like yaws patients, pinta patients are treated with penicillin.

CHANCROID

Chancroid is a venereal disease which is contracted almost exclusively through sexual intercourse. It is characterised by formation of very painful soft ulcers (hence, the other designation of the disease—soft chancre).

The causative agent of chancroid was discovered in 1885 by the Italian scientist Ferrari and, independently, in 1887 by the Russian scientist O. Petersen. It is therefore called the Ferrari-Petersen streptobacillus and sometimes incorrectly referred to as the *Hemophilus ducreyi*, after Ducrey, the scientist who described its properties in detail in 1889. It is a short rod, 1.5μ long and 0.5 μ thick. In the pus discharged from the chancroid ulcers the bacilli are attached to each other end to end in long or short chains, whence the designation "streptobacillus". The chains are sometimes arranged parallel to each other (Fig. 158).

It is believed that chancroid is contracted only if the continuity of the epidermis or of the epithelium of the mucous membranes is disrupted.

The incubation period of chancroid is not long; it usually takes 3-4 days from the moment of inoculation to the formation of the characteristic ulcer. The first signs of the developing disease can be seen already a few hours after inoculation when a small bright-red macule has formed at the site of entrance of the causative agent. On the second day this macule changes to a papule and then a pustule, which soon bursts and by the end of the third or fourth day is transformed into an ulcer.

The chancroid ulcer is irregularly shaped and is distinguished by its bright inflammatory redness. At first it varies in size from that of a millet seed to that of a lentil, but after a while reaches the size of a 15-20 kopek piece and even larger. The eroded and corroded edges of the ulcer hang over the floor which is uneven and is covered with a copious purulent discharge (Fig. 157). The ulcer is soft and very painful, especially on palpation. In this condition the ulcer persists for about 3 weeks (in the absence of treatment) and then begins to heal—the amount of purulent discharge decreases, the ulcer becomes filled with granulations and cicatrises. The healing of the ulcer takes 4-6 weeks.

The Ferrari-Petersen streptobacillus can be seen in the matter discharged from the ulcer. Exuding from the ulcer the copious purulent discharge comes in contact with other parts of the skin and mucous membranes and may cause formation of new ulcers. The chancroid ulcers are therefore multiple and may coalesce.
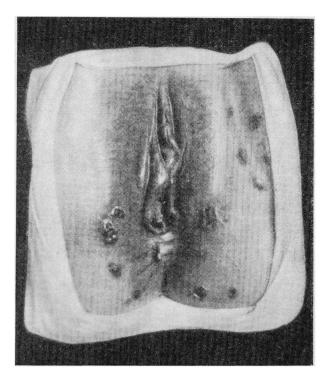
Fig. 157. Chancroid

Ulcers at different stages of development may be discovered in the selfsame patient; they may be small, newly-appeared ulcers, large ulcers at the height of disintegration, and healing, cicatrising ulcers.

Since chancroid is nearly always contracted through sexual intercourse the ulcers usually localise on the genitals. Women often develop ulcers also in the anal region. These ulcers are particularly painful, especially during defecation. In slovenly patients ulcers may also appear on the pubes, thighs and abdomen.

Chancroid may become complicated by phimosis or paraphimosis. In debilitated patients, alcoholics and persons with chronic diseases the ulcer may become gangrenous or phagedenic and wreak extensive destruction. In some cases chancroid ulcers do not heal for a long time and gradually enlarge, seem to "creep". This variety of chancroid is called serpiginous (or creeping). It is characterised by a very protracted course and large ulcers.

Chancroid ulcers always heal by leaving scars. Untreated or improperly treated chancroid is accompanied by affection of the inguinal lymph nodes in about 40 per cent of the cases. Lymph-

Fig. 158. Chancroid streptobacillus (from A. Zenin
and N. Torsuyev)
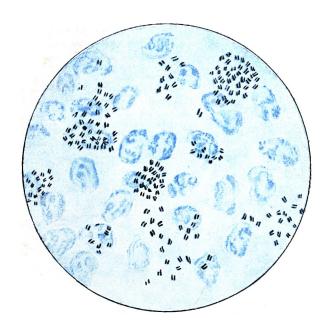
Fig. 159. Gonococci stained with methylene blue
(from A. Zenin and N. Torsuyev)
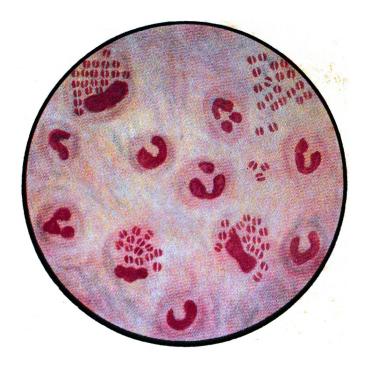
Fig. 160. Neisseria gonorrhoeae. Gram-stained
(from Predtechensky)

adenitis in chancroid patients—chancroid bubo—most commonly develops in the 3rd or 4th week of the disease. The lymph nodes enlarge and may reach the size of a plum or a chicken egg. They are very painful, soft, elastic and adhere to each other and to the surrounding tissues. The pains increase and make walking diffi-cult; the skin over the affected nodes reddens and adheres to them. Then the chancroid bubo softens and opens discharging a large amount of pus. After opening the bubo forms an ulcer. In some cases it clears up rapidly and becomes filled with granulations (simple bubo), in others the ulcer enlarges and assumes the charac-ter of a typical chancroid ulcer, its copious purulent discharge con-taining Ferrari-Petersen streptobacilli (virulent bubo).

It is very important to be able to distinguish a chancroid from a hard chancre (primary syphilitic lesion).

The following are the most important distinguishing features of these diseases.

1. The incubation period of chancroid is 2-4 days, of syphilis—3-3.5 weeks.

2. Hard chancre has a regular round or oval form, chacroid—an irregular form.

3. Hard chancre is most commonly an erosion and much less frequently a superficial ulcer, chancroid is nearly always an ulcer.

4. The edges of hard chancre are even and gently slope to the floor which is saucer-shaped; the edges of chancroid are uneven, eroded and corroded.

5. The floor of hard chancre is even and lustrous, the dischar is scant and serous; the floor of chancroid is uneven and covered with pus.

6. The borders of hard chancre are clearly defined, and the surrounding skin is not inflamed; the borders of chancroid are indistinct, and the surrounding skin is red and edematous.

7. Hard chancre is densely elastic and painless; chancroid is painful and not indurated.

8. In hard chancre the adjacent lymph nodes are enlarged, densely elastic, mobile and painless; in chancroid the inguinal lymph nodes are either unaffected or their enlargement is of an acutely inflammatory character and often ends in suppuration.

9. The discharge of hard chancre reveals in dark-field illumi-nation the *Treponema pallidum*, whereas the stained smears taken from the depth of a chancroid ulcer contains the Ferrari-Petersen streptobacillus.

Mixed chancre. Some cases are characterised by simultaneous infection with syphilis and chancroid. The chancre formed in these cases is called a mixed chancre.

In patients inoculated simultaneously with syphilis and chan-croid the disease at first runs the course of a chancroid and only several weeks later, after the incubation period of syphilis, signs

of primary syphilis come to the fore. But, if inoculation with chancroid occurred at the end of the incubation period of syphilis, after appearance of the primary lesion, the symptoms of chancroid may be camouflaged by signs of syphilis. The diagnosis is rendered the more difficult since it is hard to find the *Treponema pallidum* in the discharge of the ulcer of mixed chancre. Moreover, the serology tests in patients with mixed chancre sometimes become positive later than in patients with the usual primary syphilitic lesion. Sometimes only the development of secondary syphilis symptoms shows that the patient had mixed chancre and not chancroid.

A rule has therefore been made, according to which all chancroid patients must be kept under medical observation for 6 months. The observation consists in regular (weekly) examinations of the patient and blood tests—Wassermann and flocculation tests.

Treatment. Chancroid is treated mainly with sulfonamide preparations both internally and externally. Norsulfazol (sulfathiazole), sulfadimesine (2-para-aminobenzol sulfamido/-4,6-dimethyl piperidine) and ethasole (2-/para-aminobenzol-sulfamido/-2-ethyl-3, 4-thiodiazole) are administered in a dose of 1 g 3-4 times a day for 10 days. Locally the ulcer is dusted with streptocide powder or covered with a streptocide water paste. After 3-5 days of external application of streptocide the redness and edema disappear and the copious discharge of pus ceases. This treatment is followed by applications of a 5 per cent dermatol (bismuth subgallate) or xeroform ointment or a 2-3 per cent ammoniated mercury ointment. Treatment with sulfonamide preparations, as a rule, prevents development of buboes.

Penicillin also produces good therapeutic effects in chancroid and is administered in a dose of 50,000 u every 3 hours (a total of 1,000,000-2,000,000 u and in some cases even more). It should be remembered, however, that administration of penicillin to a patient with mixed chancre may prolong the incubation period of syphilis and thereby lead to late identification of this disease. It is therefore best to use sulfonamide preparations in the treatment of chancroid patients and resort to penicillin therapy only in cases of any contraindications or intolerance of these preparations. If chancroid buboes have formed, autohemotherapy and milk injections are resorted to.

Chancroid was a rare occurrence in the Soviet Union even before the last war, the incidence of this disease having enormously decreased compared with the prerevolutionary time.

Chancroid has been completely eliminated in the U.S.S.R., no cases of this disease having been observed since 1950.

In some countries chancroid continues to be a widespread disease. For example, Graham, American venereologist, reports that 4,813 chancroid cases were recorded in the U.S. Navy in the Far East in 1951 alone.

GONORRHEA

Gonorrhea is a venereal disease usually acquired in sexual intercourse, although it may also be communicated nonvenereally.

The disease is caused by the gonococcus discovered in 1879 by Neisser, German scientist; it belongs to the group of diplococci (paired cocci). Both cocci look like coffee beans (Figs. 159 and 160). The gonococcus is 1.6 μ long and 0.8 μ wide. Gonococci are characterised by their arrangement inside leukocytes and are noted for low resistance to the effects of temperature above 39°C and many disinfectants (silver nitrate, protargol, potassium permanganate, mercuric chloride, etc.).

The gonococcus causes the disease only in man by developing mainly in the mucous membranes coated with columnar epithelium (urethra, mucosa of the cervix uteri, conjunctiva of the eye) and less frequently in the mucous membranes coated with squamous epithelium (urinary bladder, vagina).

The gonococcus does not affect animals. It is possible to infect animals (mice) by introducing gonococci into the abdominal cavity only under laboratory conditions.

Men and women contract the disease almost exclusively in sexual intercourse, usually in casual sexual relations. Patients with chronic gonorrhea or with acute gonorrhea during the period of subsidence of the process are a source of gonorrheal infection.

Little girls become infected with gonorrhea nonvenereally from their mothers, older sisters and infected nurses. Nonvenereal inoculation with gonorrhea occurs through objects of common use—bedding, towels, chamber-pots, basins, sponges. Gaining entrance to the urethral mucosa or the mucosa of the cervix uteri the gonococcus rapidly multiplies and invades the submucous layer. From the moment the gonococcus penetrated to the mucosa to the appearance of signs of the disease usually takes 3-5 days, which is the incubation period of gonorrhea. During this period the gonococci invade the submucous tissue, on the one hand, and certain changes in the reactivity of the organism caused by multiplication of the gonococci and the effects of the toxins produced by them occur, on the other hand. The acute inflammatory process in the mucosa of the urethra may spread to the adnexa of the testes, prostate and seminal vesicles in men, and from the urethra and cervix uteri to Bartholin's glands, the uterus and its adnexa, and rectum in women.

Inadequate resistance of the organism in untreated cases may give rise to gonococcal lesions in the joints, gonococcal endocarditis and gonococcal sepsis. This indicates that gonorrhea is always a general infection of the entire organism, although most patients have inflammatory foci only in the urogenital organs.

The gonococcus may be brought to the mucous membranes of the eyes by the patient's hands contaminated with pus. This gives rise to acute gonorrheal conjunctivitis or blennorrhea; in this disease the mucous membranes of the eyes become extremely red and discharge a great deal of yellowish-greenish pus; the eyelids grow red and edematous. Pus containing gonococci may get into the eyes of a newborn during birth.

GONORRHEA IN MEN

Gonorrhea in men begins with affection of the urethra where the gonococci gain entrance during sexual intercourse with gonorrhea-infected women. The incubation period of gonorrhea may be from 1 day to 2-3 weeks, but in most cases it is 3-4 days. At the end of this period the patient begins to feel an itching and burning in the region of the external orifice of the urethra. A redness and mild edema appear around the external urethral orifice, yellowish-greenish pus exudes from the urethra, and urination is painful. If the patient is asked to urinate into two glasses (glass test), the first glass will contain turbid urine because of the presence of pus, and the second glass will contain clear urine.

In inflammation of only the anterior part of the urethra (from the external orifice to the external sphincter of the urethra) the first portion of urine washes out the accumulated pus and clean urine is voided into the second glass. The external sphincter of the urethra is always (except the moment of urination) contracted and does not pass pus into the posterior part of the urethra and the bladder.

This result of the glass test indicates affection of only the anterior part of the urethra —*anterior gonorrheal urethritis.*

Microscopic examination of smears of pus from the urethra readily reveals gonococci mainly inside leukocytes.

Without treatment urethritis may subside in 2-3 weeks. But often, especially when improperly treated, the inflammatory process spreads to the posterior part of the urethra with the result that *posterior gonorrheal urethritis* develops. The patient has frequent painful urges to urinate. The pain increases particularly at the end of urination, the last drops of urine often being stained with blood. The glass test shows both portions of the urine to be equally turbid. In inflammation of the posterior part of the urethra the pus cannot penetrate through the contracted external urethral sphincter to the anterior part of the urethra, but easily penetrates

into the bladder through the much weaker internal urethral sphincter. Owing to the passage of pus into the urinary bladder the urine mixes with the pus and becomes uniformly turbid.

The inflammatory process always involves the mucosa of the internal urethral sphincter and the surrounding part of the mucosa of the fundus vesicae. The patient develops frequent urges to urinate and an intense pain, especially at the end of urination on contraction of the internal urethral sphincter. The last drops of urine contain blood because on contraction of the internal sphincter at the end of urination the sharply inflamed mucosa of this part of the urethra begins to bleed.

Posterior urethritis is often complicated by gonorrheal inflammation of the prostate, adnexa of the testes, seminal vesicles and Cowper's glands.

If the gonorrhea patient is untreated or is treated irregularly or improperly, the urethritis is not cured after subsidence of the acute inflammatory phenomena, but becomes chronic.

Chronic gonorrheal urethritis is characterised by mild pain. The chronic inflammatory process persists mainly in the mucous glands of the urethra and in various infiltrated parts of the submucous tissue of the urethra. It is manifested only in a small drop of mucopurulent discharge from the urethra, which can be observed in the morning before urination. The first portion of urine in the glass test of these patients contains purulent and mucopurulent filaments and flakes suspended in transparent urine. Microscopic examination of these filaments and flakes may reveal gonococci. The second portion of urine usually looks normal. Patients often complain of an itching, tickling and, sometimes, burning sensation in the urethra.

Untreated chronic urethritis may last very long. Under the influence of various stimuli—sexual intercourse, alcoholic drinks, etc.—chronic gonorrheal urethritis may become aggravated, in which case discharge from the urethra again becomes copious and purulent, urination is accompanied by colic, and the urine of the first portion becomes turbid. However, these phenomena soon subside and disappear without any treatment, and urethritis becomes chronic again.

Chronic urethritis often combines with gonorrheal affection of the prostate and the seminal vesicles.

In some cases acute gonorrheal urethritis is from the very outset characterised by mild inflammatory phenomena. During urination there is either little colic or no colic at all. The external orifice of the urethra shows but scant purulent, mucopurulent or even mucous discharge. The urine is but slightly turbid in the first portion and is clear in the second portion. This form of the disease is called sluggish or recent torpid gonorrhea. The cause of such a course of acute gonorrhea lies in the reduced reactivity of the

patient's organism.Some role is undoubtedly also played by a change in the properties of the gonococci. Cases of sluggish gonorrhea have become much more frequent since the introduction of sulfonamide preparations and penicillin into the therapy of the disease. Gonococci insensitive to sulfonamide preparations and scarcely sensitive to penicillin are often found in patients with recent torpid gonorrhea. Sluggish gonorrhea is very difficult to diagnose because of the absence of complaints of pain and of discharge, and the absence of clearly marked inflammatory phenomena. In such patients it is often also difficult to find gonococci in the discharge from the urethra. It is sometimes necessary to take repeated smears with a blunt curet from the walls of the urethra to discover gonococci.

Recent torpid gonorrhea is particularly unpleasant and dangerous since the patients are often unaware of their condition and continue to partake of alcoholic drinks and have sexual intercourse. This leads to infection of women with gonorrhea and is conducive to development of various complications of gonorrhea in the patients themselves Complications are much more frequently observed in sluggish gonorrhea than in usual acute gonorrhea.

Complications of gonorrhea in men. The extension of the gonorrheal process from the urethra to other organs—the prostate, adnexa of the testes, the seminal vesicles, Cowper's glands, urethral glands and paraurethral ducts—is called gonorrheal complications.

The development of these complications is due to a number of causes. The main causes are: lowered resistance of the patient's organism, unfavourable external influences, inadequate or improper and irregular treatment, and the properties of the gonococcus itself.

The lowered resistance of the organism may be due to disturbances in the functions of the nervous system, internal diseases, overstrain, unwonted hard work, sexual excitement, and use of alcoholic beverages.

Gonorrheal complications develop in patients with both the acute and chronic forms of gonorrhea. Gonorrheal complications also include balanoposthitis, phimosis and paraphimosis.

Gonorrheal inflammation of urethral glands. Some patients with gonorrheal urethritis develop inflammation of the mucous glands of the urethra. Examination of the urethra by means of a urethroscope reveals dilated, gaping ostia of the urethral glands and an areola of redness and infiltration.

In the glass test the first portion of urine of such patients often contains purulent filaments and flakes.

Gonorrheal prostatitis. Inflammation of the prostate gland, or prostatitis, is one of the most common complications of gonorrhea. Penicillin has made it a rare complication of acute gonorrhea. Prostatitis is observed much more frequently in patients with chronic gonorrhea. It is also often observed in patients with sluggish gonorrhea.

Prostatitis may be acute or chronic. The following forms of acute prostatitis are distinguished: (1) *catarrhal* in which the inflammatory process involves mainly the excretory ducts of the glandular lobules of the prostate, (2) *follicular*, characterised by involvement of various whole lobules of the gland in the process, and (3) *parenchymatous* in which both the interstitial and glandular tissues are affected by the acute inflammatory process.

Catarrhal prostatitis causes no pain and palpated through the rectum the prostate gland appears unaffected.

Patients with follicular prostatitis may have complaints of frequent painful urination; on palpation the prostate gland appears enlarged, painful and with an uneven surface.

Parenchymatous prostatitis causes considerable disturbances in the patient's general conditions: fever, pains during defecation and retention of urine. Palpation reveals a sharp enlargement of all or part of the prostate, compactness and tension of its tissue and intense pain. Formation of an abscess in the prostate gland is also possible.

Chronic prostatitis is observed in patients with chronic gonorrhea. The patients complain of discomfort and pain in the anal region and the perineum, impaired sexual ability, and frequent urination. Palpation through the rectum shows the prostate to be either of its usual size or enlarged, compact or, on the contrary, flabby, uneven or unusually smooth.

Gonorrheal epididymitis or inflammation of the adnexa of the testis usually develops in patients with acute gonorrhea or with aggravation of chronic gonorrhea. Penicillin therapy has made epididymitis a rare gonorrheal complication. The disease most commonly develops in patients who violate the prescribed regimen, for example, have sexual intercourse, consume alcoholic beverages, exert unwonted physical effort.

In most cases gonorrheal epididymitis develops acutely with pain in the inguinal region and a testis, and a rise in temperature. Within a few hours, sometimes the next day, the affected epididymis becomes enlarged, indurated and painful on palpation. The testis is also somewhat enlarged and painful. The spermatic cord is thickened, indurated and painful. The skin of the scrotum becomes red and edematous. On development of epididymitis the symptoms of gonorrheal urethritis diminish, the purulent discharge from the urethra and the pain on urination decrease or disappear.

On healing epididymitis always leaves a scar in the epididymis, which is likely to cause obstruction of the ejaculatory duct. In most cases bilateral epididymitis results in sterility.

Gonorrheal vesiculitis or inflammation of the seminal vesicles often occurs simultaneously with epididymitis or prostatitis and may also be acute or chronic. The complaints of vesiculitis patients are similar to those of prostatitis patients. The patients are some-

times discomforted by painful pollutions (seminal discharge) and blood in the semen. Patients with chronic vesiculitis often have disorders of the sexual function.

Gonorrheal cowperitis. Affection of Cowper's glands by the gonococcus—gonorrheal cowperitis—is a less frequent gonorrheal complication and may also be acute or chronic.

In the catarrhal form of cowperitis the inflammatory process involves mainly the excretory ducts of Cowper's glands. The follicular form affects not only the excretory ducts but also various gland lobules. Neither of these forms of cowperitis causes any subjective sensations in the patient. In some cases the inflammatory process involves the entire tissue of the gland (parenchymatous cowperitis) or even spreads to surrounding tissues (paracowperitis). These cases are characterised by pain in the perineum, the pain increasing during defecation, sitting on something hard, and upon movement.

Gonorrheal paraurethritis, or inflammation of paraurethral ducts, may arise in the very beginning of gonorrheal infection or may develop later as a result of penetration of pus from the urethra to the orifice of a paraurethral duct.

The paraurethral ducts are narrow, blind canals, sometimes 6-7 cm long. They open only on the glans penis, the spongy part of the urethra, the urethral groove and the skin of the penis.

Gonorrheal paraurethritis may not cause any subjective sensations in the patient. Pressure on the walls of a paraurethral duct results in discharge of a purulent drop which may contain gonococci. Uncured paraurethritis may cause relapses of gonorrheal urethritis.

Stricture of the urethra. Inflammatory infiltrates very often develop in the submucous tissue of patients with acute or chronic gonorrheal urethritis. In some cases the infiltrate is not resorbed but is replaced by cicatricial tissue which may compress the urethra and constrict its lumen. Cicatricial constriction, or stricture of the urethra, makes urination somewhat difficult, depending on the extent to which the lumen of the urethra is constricted. When the urethra is constricted the stream of urine becomes thin, and falls plumb down; in such cases it takes longer to urinate. At the end of urination the urine continues for some time to fall in drops. In time the constriction may progress. In such patients the urine is sometimes voided only in drops.

Due to constant tension the muscles of the bladder weaken, and, on urination, the bladder is not drained completely (residual urine). This leads to inflammation of the bladder, ureters and kidney pelves. The first signs of a developing stricture usually appear in the patient several years after an attack of gonorrhea.

Gonorrheal cystitis. Inflammation of the bladder is one of the less common complications of gonorrheal urethritis.

In gonorrhea patients cystitis is in most cases produced not by the gonococcus, but by the colon bacillus, the streptococcus and staphylococcus. In some cases these microbes are brought into the bladder as a result of violation of the rules of asepsis during treatment. In other cases cystitis develops in patients with urethral stricture as a result of retention of urine.
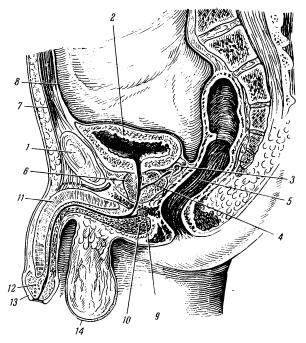
Fig. 161. Male pelvis (from A. Kartamyshev)

1—symphysis pubis; 2—bladder; 3—seminal vesicle; 4—ejaculatory duct; 5—rectum; 6—prostate; 7—abdominal wall; 8—peritoneum; 9—Cowper's gland; 10-12—corpora cavernosa of the urethra and penis; 13—prepuce; 14—scrotum.

Cystitis patients complain of frequent urination with irrepressible painful urges. The urination is very painful, especially at the end. The urine becomes turbid because it contains pus, and often red because it is stained with blood. General indisposition is frequently observed: poor sleep, lack of appetite, weakness, and irritability.

GONORRHEA IN WOMEN

The clinical picture and course of gonorrhea in women greatly differ from those observed in men, which is due to the differences in the anatomy and physiology of the male and female genitalia (Figs. 161 and 162).

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In women not only chronic but acute gonorrhea often causes negligible subjective sensations and feebly marked objective symptoms. That is why the woman who has become infected with gonorrhea often long fails to notice the developing disease. Sometimes she finds out about her illness only in a medical institution
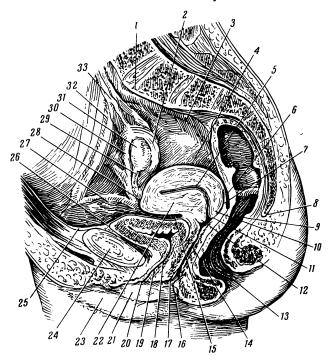
Fig. 162. Female pelvis (from A. Kartamyshev)

1—sacral projection; 2 and 30—fallopian tube; 3—peritoneum; 4, 22 and 28—uterus; 5—os uteri internum; 6 and 15—pouches of Douglass; 7—posterior fornix; 8—coccyx; 9—rectum; 10 and 12—cervix uteri; 13—anus; 14—vagina; 16 and 17—hymen and introitus vaginae; 18, 19, 20—urethra; 21—labium minus; 23—labium majus; 24—symphysis; 25—median umbilical ligament; 26—bladder; 27, 29 and 33—uterine ligaments; 31—ovary; 32—iliac vein.

where she is summoned for examination as the source of gonorrheal infection.

In woman gonorrhea in most cases affects at once several organs from the very outset: the urethra, paraurethral ducts, Bartholin's glands and the cervix uteri. Even if only the cervix uteri is affected in the beginning of the disease, the purulent discharges containing gonococci easily get into the urethra, the paraurethral ducts, Bartholin's glands and the rectum and cause their infection. The gonorrheal process in woman is therefore nearly always of a multifocal character. Gonorrhea in woman is called uncomplicated,

if only the lower division of the urogenital organs (urethra, para-urethral ducts, cervix uteri and Bartholin's glands) is affected, and complicated or ascending, if the gonococcus has penetrated through the internal os uteri and has caused infection of the cavity, adnexa and ligaments of the uterus, periuterine cellular tissue and the peritoneum of the minor pelvis.

Gonorrhea in woman may run an acute or chronic course.

Uncomplicated Gonorrhea in Women

Gonorrheal endocervicitis. Gonorrheal inflammation of the lining membrane of the canal of the cervix uteri, or endocervicitis, is observed in nearly all women infected with gonorrhea. A purulent vaginal discharge which often irritates the mucous membranes of the external genitalia and the surrounding skin ("corrosive leukorrhea") is one of the signs of acute endocervicitis.

Sometimes the patients have a sensation of heaviness in the pelvis minor and pains in the sacrum and small of the back. Very often there are no subjective sensations, the woman attaches no importance to the leukorrhea and does not apply for medical aid.

Examination of the cervix uteri with a vaginal speculum shows the cervix to be red, loose and edematous. A bright-red, irregularly shaped erosion may often be seen around the external os uteri. A mucopurulent discharge in which gonococci may be seen under the microscope runs down in the form of a long ribbon from the external os. After some time the inflammatory process subsides. If the patient is not treated, acute endocervicitis becomes chronic.

Chronic endocervicitis causes no subjective sensations. The discharge from the cervix uteri becomes scant. Examination with a vaginal speculum reveals a small erosion, sometimes with a granular surface, around the external os of the cervix uteri. In some cases the cervix becomes enlarged and indurated because of the extension of the inflammatory process to the muscular layer of the uterus. The cervix uteri turns purplish. Sexual intercourse, hard physical work and consumption of alcoholic beverages may aggravate the course of chronic endocervicitis. There is always a danger that endocervicitis may develop into ascending gonorrhea.

Gonorrheal urethritis. Gonorrheal inflammation of the urethra is also one of the most common manifestations of gonorrhea in women. Acute gonorrheal urethritis develops 3-4 days after inoculation with symptoms of moderate pain and burning during urination. Sometimes urination is painless.

In many cases the patient does not notice these symptoms or attaches no importance to them and does not consult a physician.

Examination of the patient with acute urethritis reveals a redness and edema around the external orifice of the urethra and

discharge of pus. The microscope shows gonococci in the smear of pus.

Chronic urethritis causes no painful sensations in women. The discharge from the urethra is of a mucopurulent character and scant. Palpation of the urethra often reveals a rather dense infiltrate in its walls. Gonorrheal urethritis is often accompanied by paraurethritis.

Gonorrheal bartholinitis. Gonorrheal inflammation of Bartholin's glands is observed in 20-25 per cent of all women infected with gonorrhea and is usually bilateral. In some cases the inflammatory process involves only the ostia of the excretory ducts, in other cases—the excretory ducts from beginning to end, and in still others—the body of the gland. In some cases Bartholin's glands develop abscesses.

Gonorrheal inflammation of the external genitalia (vulvitis) and of the vagina (vaginitis) occurs rarely and is observed only in pregnancy and during the climacteric. Normally the mucosa of the external genitalia and the vagina offers unfavourable conditions for the development of the gonococcus; the gonococcus may develop only if these conditions are altered by the reorganisation taking place in the organism during pregnancy and the climacteric, after surgical removal of the ovaries, etc.

Ascending Gonorrhea

In untreated or improperly treated cases uncomplicated gonorrhea in women develops into complicated or ascending gonorrhea. The development of ascending gonorrhea is also due to lowered resistance of the organism, which is favoured by disturbances in the function of the nervous system and diseases of internal organs.

Various violations of the gonorrhea patient's regimen—sexual intercourse and even sexual excitement, hard physical work, overstrain, overcooling, consumption of alcoholic beverages—often serve as the impetus for the extension of gonorrhea to the uterus and its adnexa. The menstrual period connected with an afflux of blood to the uterus and its adnexa, a massive desquamation of the uterine epithelium and opening of the internal os also favours development of complicated gonorrhea.

In ascending gonorrhea the patient has pains, sometimes very intense, in the lower part of the abdomen and in the small of the back, often of a paroxysmal character. The discharge from the cervix uteri becomes copious, thin and seropurulent; sometimes it contains blood; the temperature often rises to 39-40°C and the patient develops chills. In some patients the gonorrheal process in the uterine adnexa causes irritation of the pelvic peritoneum owing to which the pains in the lower part of the abdomen increase, the muscular wall of the abdomen is tense, and nausea, vomiting

and constipation appear. In other patients ascending gonorrhea develops sluggishly, without sharp pains and clear clinical signs.

The localisation of the inflammatory process in ascending gonorrhea is ascertained by a bimanual examination which must be performed very cautiously.

In *gonorrheal endometritis* the uterus is enlarged, somewhat indurated, and painful. The menses often occur ahead of time, are prolonged and sometimes very copious.

In gonorrheal *adnexitis* it is not always possible to determine the localisation of the inflammatory process by palpation. On internal examination the catarrhal inflammatory process in the fallopian tubes is characterised only by pain. In the presence of infiltrate in the submucosa of the fallopian tubes and accumulation of serous or purulent exudate a painful formation varying in density and size may be palpated in the region of the adnexa.

Gonorrheal adnexitis is often bilateral and may become chronic. Patients with chronic adnexitis have no sharp pains or acute inflammatory phenomena. They complain of recurrent dull pains in the lower part of the abdomen and in the small of the back, leukorrhea and disturbances in the menses. Chronic adnexitis may become periodically aggravated under the influence of sexual intercourse, hard physical work, psychic trauma, colds and excessive use of alcoholic beverages. Chronic gonorrheal adnexitis is often accompanied by irritability, rapid tiring and reduced working capacity.

In many cases bilateral gonorrheal adnexitis leads to sterility by obstructing the fallopian tubes as a result of an adhesive inflammatory process in the tubes or formation of cicatricial tissue. Women infected with chronic gonorrhea constitute a serious hazard as regards spreading gonorrhea, especially if they live a disorderly sexual life.

Gonorrheal proctitis. Gonorrheal inflammation of the rectum, or proctitis, is observed almost exclusively in women and develops as a result of an inflow of gonococcus-containing pus from the vagina. The patient may also carry the purulent discharge from the vagina to the anal mucosa by washing or wiping the perineal or anal region. Patients with gonorrheal proctitis show a purulent discharge in the folds of the anal mucosa and sometimes a redness around the anus. In some cases painful fissures are formed.

The patients complain of itching and slight pains in the anus, and sometimes sharper pains during defecation. Proctitis may cause no subjective sensations at all.

GONORRHEA IN GIRLS

Girls contract gonorrhea nonvenereally, usually from women infected with gonorrhea—mothers, sisters, nurses and other women with whom they are in close contact. The inoculation most com-

monly occurs through using the chamber-pot, wash basin, sponge or towel used by a gonorrhea-infected woman. It may often also occur through sleeping in one bed with the gonorrhea infected mother or other woman.

Gonorrhea is contracted by girls of any age, but most commonly between 3 and 7 years of age.

Gonorrhea in girls considerably differs from gonorrhea in women. *Gonorrheal vulvovaginitis*—inflammation of the mucous membranes of the external genitalia in the region of the ostium vaginae and of the vaginal mucosa—is the most frequent gonococcal affection in girls.

In adults vulvitis and vaginitis are rare occurrences because of the differences in the structure of the epithelium of the mucous membranes and of the chemical composition of their discharge in women and girls.

In gonorrheal vulvovaginitis the mucosa and skin of the external genitalia become red and edematous. A purulent discharge exudes from the vagina. The mucosa of the introitus is bright-red and infiltrated. Gonorrheal urethritis very often develops simultaneously with vulvovaginitis or soon afterwards.

The mucous membranes around the urethral orifice and the clitoris become bright-red and edematous. Pus is discharged from the external urethral orifice. Urination causes pain and a sensation of burning and is more frequent than usual.

The copious purulent discharge from the vagina leads to irritation of the skin of the labia majora, the inguinal folds, medial surface of the thighs and the perineum. The skin on these parts becomes red, sometimes slightly edematous and covered with erosions and purulent crusts.

The condition is often accompanied by development of *gonorrheal proctitis*—gonorrheal inflammation of the rectal mucosa. A redness appears around the anus, the folds of the mucosa become swollen, and in some cases fissures are formed. A purulent or mucopurulent discharge exudes from the anus, and defecation may become painful.

Gonococci are found in the discharge from the urethra, vagina and rectum. Smears to be examined for gonococci should be taken with particular caution by means of a blunt curet.

In girls gonorrhea may also affect the mucosa of the canal of the cervix uteri.

Ascending gonorrhea is extremely rarely observed in girls.

The general state of the organism of girls infected with gonorrhea is disturbed: the children become sluggish and irritable and refuse food. Within 2-3 weeks the acute phenomena begin to abate: the purulent discharge from the vagina and urethra decreases, urination becomes normal, and the irritation of the surrounding skin disappears.

In some cases acute gonorrhea in girls runs a sluggish course from the very outset: the subjective sensations are negligible, the discharge is scant, the redness and edema are but feebly marked.

If the treatment of acute gonorrhea in girls is not started in due time or is administered improperly, no cure is effected and the disease becomes chronic.

In girls infected with chronic gonorrhea the mucous membranes are but mildly inflamed.

There is but a circumscribed and slight redness around the external urethral orifice and ostium vaginae. The vaginal and urethral discharge is scant and mucopurulent. Gonococci are not always found in the smears taken from the vaginal, urethral and anal discharge.

Chronic gonorrhea in girls may long remain concealed, but under the influence of the changes taking place in the organism, for example, as a result of various infections (measles, scarlet fever, etc.), colds, etc., the disease may from time to time become aggravated. In such cases the picture of the disease resembles that of acute gonorrhea, but the acute phenomena soon subside even without treatment.

TREATMENT OF GONORRHEA

Gonorrhea is a general infectious disease with predominant localisation of the pathologic foci in the urogenital organs. That is why methods of general and local therapy are used in the treatment of gonorrhea.

General treatment. Penicillin therapy is the basic method of general treatment of gonorrhea today. The use of penicillin makes it possible to cure most gonorrhea patients in short periods of time.

Penicillin is administered intramuscularly in a dose of 50,000-100,000 u dissolved in 3-4 ml of sterile physiologic solution every 3 hours, or in the form of ecmonovocillin in a dose of 600,000 u once a day or 300,000 u twice a day.

In acute uncomplicated gonorrhea in men and women a dose of 600,000 u of penicillin or ecmonovocillin is prescribed. For men with acute complicated gonorrhea, for women with ascending gonorrhea and for men and women with chronic gonorrhea the course dose of penicillin must be increased, according to the pathologic process, to 1,000,000-3,000,000 u.

Gonorrhea-infected girls are prescribed the same doses of penicillin as adults.

For ambulant gonorrhea patients it is best to use ecmonovocillin or bicillin.

In acute torpid gonorrhea and in acute complicated and chronic gonorrhea in men and women the doses of ecmonovocillin are increased, as in the treatment with penicillin, to 1,000,000-

3,000,000 u, the preparation being administered in a dose of 600,000 u once a day or 300,000 u twice a day.

For ambulant gonorrhea patients penicillin may be administered in the patient's own blood. To administer penicillin in the patient's own blood, 300,000 u of the preparation are first dissolved in 1 ml of sterile physiologic solution and the resultant solution is then mixed with 5 ml of blood taken from the patient's vein. Penicillin in the patient's own blood is injected twice a day.

Bicillin-1 and bicillin-3 are also used in the treatment of gonorrhea patients and are administered once a day in a dose of 600,000 u.

If penicillin preparations cannot be administered in the form of injections, the patient is prescribed phenoxymethyl-penicillin per os. This preparation is taken half an hour before meals in a dose of 200,000 u 3-4 times a day. A course of treatment requires 2,000,000 u for patients with acute uncomplicated gonorrhea and 4,000,000-6,000,000 u for patients with acute complicated, recent torpid and chronic gonorrhea.

In most cases gonorrhea patients treated with injections of penicillin or ecmonovocillin are simultaneously given sulfonamide preparations—15 g of norsulfazol, ethasole or sulfodimesine—per os (p. 307).

In cases of acute complicated and chronic gonorrhea it is necessary, in addition to administration of penicillin, to employ immunobiologic methods of therapy and administer local treatment.

In gonorrhea penicillin produces potent therapeutic effects. The gonococci disappear from the discharge in the first 4-6 hours after the beginning of penicillin therapy. The discharge decreases and becomes mucopurulent. The inflammatory phenomena in the urethra disappear a few days after the beginning of the treatment. Gonorrheal complications rarely develop if penicillin is administered from the very beginning of the disease.

According to I. Porudominsky, complications developed only in 0.5-1 per cent of the cases of acute uncomplicated gonorrhea in men treated with penicillin.

Streptomycin has of late been successfully used in the treatment of gonorrhea patients. This antibiotic is particularly indicated for the treatment of patients but temporarily relieved by penicillin therapy.

Streptomycin is administered intramuscularly in a dose of 0.5 g in 5 ml of sterile physiologic solution twice a day. Patients (men and women alike) with acute uncomplicated gonorrhea are administered 1 g of streptomycin per course of treatment. For the treatment of men and women with acute complicated, chronic and recent torpid gonorrhea the course dose of streptomycin is

increased to 2-4 g. In cases of acute complicated and chronic gonorrhea this treatment is supplemented by local therapy.

Streptomycin is now comparatively rarely used in the treatment of gonorrhea because the gonococcus has become much less sensitive to it.

Synthomycin (chloramphenicol) is another agent used for the treatment of gonorrhea. This preparation is administered perorally in a dose of 10 g per course of treatment to patients with uncomplicated gonorrhea. In cases of complicated gonorrhea in men, ascending gonorrhea in women, chronic and recent torpid gonorrhea the course dose of synthomycin is 12-15 g.

Synthomycin is administered in a dose of 0.5 g 4 times a day for 5-8 days. The nocturnal interruption in the administration of the preparation must not exceed 7-8 hours.

The gonococci disappear from the discharge during the first 4-5 hours of synthomycin treatment.

Synthomycin produces good effects in the treatment of gonorrhea in girls. The single dose of the preparation is calculated at 0.02 g per 1 kg of the patient's weight and is administered 5 times a day with 4-hour intervals during the day and a 7-hour interruption for the night. The course dose is prescribed according to age and varies from 7-8 to 10 g.

Levomycetin or chloromycetin which is closely related to synthomycin is a more active preparation for the treatment of gonorrhea. This preparation is administered in a dose of 0.5 g 6 times on the first day and 0.5 g 4 times a day during the subsequent days. A course of treatment requires 5 g for acute uncomplicated gonorrhea and 7 g for acute complicated, recent torpid and chronic gonorrhea.

Girls are given levomycetin in a dose of 0.25 g 4 times a day, 4-5 g per course of treatment.

Biomycin (chlortetracycline), tetracycline and terramycin are also productive of good effects in the treatment of gonorrhea. These antibiotics are usually administered in a dose of 0.2 g 5 times a day, 3-4 g per course for acute uncomplicated gonorrhea and 5-7 g per course for complicated, chronic and recent torpid gonorrhea.

Sulfonamide preparations played an important part in the treatment of gonorrhea patients before the appearance of penicillin and other antibiotics. Today these preparations are only of auxiliary importance in the treatment of gonorrhea patients since their effects are very much inferior to those produced by antibiotics.

Sulfonamide preparations are administered mainly together with penicillin or streptomycin. Norsulfazole, ethasole and sulfodimesine are given per os in a dose of 1 g 5 times a day for 3-4 days, a total of 15-20 g per course of treatment.

Immunotherapy (serotherapy) plays an important role in the treatment of patients with complicated and, especially, chronic

gonorrhea. Development of gonorrheal complications and the change of gonorrhea to the chronic form must be considered a sign of lowered resistance of the organism and inadequate development of the processes of immunity. That is why such effective therapeutic agents as penicillin and other antibiotics are not always productive of positive effects in the treatment of patients with complicated, recent torpid and chronic gonorrhea.

To increase the reactivity of the organism and strengthen the immune processes in gonorrhea patients immunobiologic methods of treatment—intramuscular injections of gonococcal vaccine and milk, and autohemotherapy—are used. The first dose of gonococcal vaccine contains 150,000,000-300,000,000 microbial bodies. Subsequently the dose is increased each time 150,000,000-300,000,000 microbial bodies.

The vaccine is administered once in 3-5 days in accordance with the reaction to the preceding injection (headache, rise in temperature, increased discharge, pain at the site of injection). The stronger the reaction to the preceding injection, the longer the interval that must be allowed to elapse before the next injection. A total of 4-8 injections is made.

Local treatment. When patients with acute uncomplicated gonorrhea are treated with penicillin, streptomycin or synthomycin, there is no need in local therapy.

Local treatment is necessary in all cases of acute complicated, chronic and torpid acute gonorrhea.

The methods of local treatment depend on the localisation and character of the inflammatory process.

For the treatment of urethritis the urethra is *irrigated* with a large amount of a warm (38-40°C) potassium permanganate solution (1 : 10,000, Janet's irrigation). As the acute phenomena abate the concentration of the solution may be increased to 1: 6,000.

The irrigation is performed once a day by means of an Esmarch's can with a rubber tube and glass or rubber nozzle. The can is placed at a height of 1-1.5 m above the level of the genitalia. The nozzle is sterilised by boiling each time it is used. The irrigation begins with disinfection of the skin around the external urethral orifice by means of a piece of cotton soaked in a 1 : 6,000 mercuric oxycyanide solution or a stream of the solution from the Esmarch's can. Then the nozzle is introduced into the urethral orifice and the solution is allowed to flow in until the anterior part of the urethra is filled, following which the nozzle is withdrawn and the solution is allowed to flow out of the urethra. This administration and draining of the solution is repeated several times, for which about 0.5 l of the solution is used. Only the anterior part of the urethra is irrigated during the first 3-5 days, after which its posterior part is irrigated. After irrigation of the anterior part the nozzle is inserted in the urethra and the solution is allowed to

flow into the urinary bladder. As the bladder fills, the patient develops an urge for urination. At this point the nozzle is withdrawn and the patient voids the irrigation solution. The filling of the bladder is repeated until the outflowing solution ceases to be turbid and retains its pink colouring, which indicates that the purulent discharge has been washed out of the entire urethra. Usually the bladder has to be filled twice.

In the treatment of urethritis complicated by affections of the mucosa of the urethral glands or formation of a soft infiltrate the patients are given *instillations* of a 0.25-1 per cent silver nitrate solution or a 1-2 per cent protargol solution.

The instillations consist in administration of 6-8 ml of one of these solutions into the anterior part of the urethra by means of a special instillator or a syringe with a rubber nozzle. The patient retains the solution in the urethra for 2-3 minutes.

Into the posterior urethra these solutions are instilled by means of a special instillator or a thin rubber catheter; this procedure must be carried out under strictly sterile conditions. After introduction of the instillator or catheter 3 ml of a 1 per cent silver nitrate solution is administered with a syringe through the catheter or instillator. The patient must not urinate for 20-30 minutes after the instillation.

Patients with chronic urethritis are sometimes administered a *bouginage*. A sterilised straight or angled bougie is introduced into the patient's urethra (the patient in a recumbent position) and left there for 5-10-15 minutes. After removal of the bougie the urethra is irrigated with a 1 : 6,000 mercuric oxycyanide solution.

A bouginage is prescribed if the patient with chronic urethritis has inflammation of the urethral glands, a hard infiltrate, or a soft infiltrate which resists treatment with instillations of silver nitrate solution. The bougie is used once in 2-3 days.

In cases of prostatitis and vesiculitis in the acute stage the patients are given hot microclysters (40-45°C); for this procedure they lie on the right side, the legs flexed in the knees. Half a glassful of water is administered into the rectum by means of a rubber syringe, and the patient is instructed to retain the water for 15 minutes.

Patients with chronic prostatitis and vesiculitis are treated with diathermy. The active electrode is introduced into the rectum, the passive electrode is placed on the perineum or above the pubes. Diathermy sessions are conducted daily.

An important part in the local treatment of patients with subacute and chronic prostatitis and vesiculitis is played by massage of the prostate gland and the seminal vesicles. The massage of the prostate must be made every other day and must consist only of superficial stroking from the periphery toward the centre. The

seminal vesicles are massaged in the direction of their excretory ducts.

After massage of the prostate, the seminal vesicles and Cowper's glands the patient voids the urine, and the urethra is irrigated with a mercuric oxycyanide or potassium permanganate solution.

Patients with acute epididymitis are prescribed moist heat in the form of hot compresses. After elimination of the acute phenomena the patients are given hot baths, paraffin therapy and diathermy. During the acute period the patients are kept in bed. Subsequently they are allowed to get out of bed, but are advised to wear a *suspensory*.

Urethritis in women is treated with irrigations (Janet's method) and instillations. The affected paraurethral ducts are cauterised by diathermocoagulation or silver nitrate on a thin probe. In patients with chronic urethritis the urethral mucosa is coated with pure ichthyol after preliminary irrigation.

Patients with endocervicitis are first given a lavage of the vagina with a warm potassium permanganate solution, after which Cusco's speculum is introduced into the vagina and the latter is filled for 5 minutes with a warm 2-3 per cent protargol solution so that it covers the cervix uteri. After this "vaginal bath" the mucosa of the cervix uteri is painted with Lugol's solution.

Patients with bartholinitis and proctitis are given hot sitz baths with potassium permanganate solution. In proctitides 5-10 ml of a 3 per cent protargol solution is administered into the anus by means of a syringe. The anal fissures are painted with a 5 per cent silver nitrate solution.

Local treatment of patients with gonorrheal endometritis and adnexitis does not differ from the treatment of these affections of other etiology; during the acute period the patients are kept in bed and are given cold applications; during the subacute period hot compresses are applied to the abdomen and the patients are prescribed the quartz lamp. Diathermy, pelotherapy and paraffin therapy are used in chronic forms. The local treatment of patients with gonorrheal arthritis is the same as in acute arthritides of other etiology, i.e., heat in the form of hot compresses and hot baths, and after termination of the acute phenomena—diathermy, pelotherapy, massage and kinesiatrics.

Treatment of girls infected with acute gonorrhea requires bed rest. Local treatment during the acute period is administered in the form of warm sitz baths with potassium permanganate (1 : 10,000) or camomile decoction. After the bath the external genitalia are carefully dried and powdered with talcum. As soon as the acute phenomena have abated vaginal lavage is begun with a potassium permanganate solution through a thin rubber catheter which is introduced into the vaginal orifice. The lavage is followed

by instillation of 3-5 ml of a 1-2 per cent protargol solution. Into the urethra 3-4 drops of a 1-2 per cent protargol solution are instilled by an eye-dropper.

If the girl also has proctitis, 5-10 ml of a 1-2 per cent protargol solution is administered into the rectum by means of a rubber syringe.

The regimen of the gonorrhea patient must foster the quickest possible cure. The patient must avoid overstrain, unnecessary worries and colds, and must have enough rest, sleep and fresh air. The patient may harmlessly do his habitual physical work. Harm is likely to be inflicted by any new and unwonted physical efforts, especially lifting weights, jumping, and riding a bicycle and motorcycle, which may lead to complications of gonorrhea in men and women.

Sexual excitement and alcoholic beverages are even more conducive to development of complications in gonorrhea patients. Patients with acute gonorrhea should be prohibited seasoned and salty foods.

Criterion of Cure in Gonorrhea

If the general and local treatment of the patient leads to disappearance of the inflammatory phenomena, the question—is the patient cured of gonorrhea—arises. This question is particularly difficult to answer if the patient has retained mild inflammatory phenomena—a slight mucous or mucopurulent discharge from the urethra, or the first portion of his urine contains filaments and flakes. In women a mucopurulent discharge sometimes long persists from the cervix uteri. In some cases these phenomena may be manifestations of chronic gonorrhea, in others—the result of an inflammatory process unassociated with the gonococcus.

Prolonged irritation of the urethral mucosa by the inflammatory process and the additional irritation by the medicinal substances may maintain the inflammatory process, although there are no more gonococci in the tissues. Such an inflammatory process is called *postgonorrheal*. To make sure the patient is cured, the latter is followed up after the treatment.

Patients treated for acute uncomplicated gonorrhea must be followed up for 7-10 days during which discharge smears are taken from the urethra, cervix uteri and other organs. Patients treated for acute complicated gonorrhea must be followed up for a longer time—2-4 weeks.

If no signs of gonorrhea appear and no gonococci are found in the smears during the follow-up, a provocation is resorted to.

In the case of men it is usually a combined provocation: (1) a bougie is introduced for 10-15 minutes or 6-8 ml of a 5 per cent silver nitrate solution is instilled into the anterior part of the urethra, (2) the patient is given an injection of 500,000,000-1,000,000,000

microbial bodies of a gonovaccine or 5 ml of milk, and (3) the patient is asked to drink 0.5-1 litre of beer. After the provocation the patient is examined over a period of 3 days, smears of his discharge taken daily. If no signs of gonorrhea are discovered, the patient is excused for a month, after which the examination and provocation are repeated. If the results of the second provocation are favourable, the patient is considered cured and is taken off the records.

Men with chronic gonorrhea must be followed up for at least 2 months. Once a week the patients must report to a medical institution for examination during which their external genitalia are inspected, smears from the urethra are taken, the glass test is performed, and the prostate gland, the seminal vesicles and Cowper's glands are examined through the rectum. If necessary the prostate is examined and a urethroscopy is made. About 1 month after such follow-up the patient is subjected to a provocation. If the results are favourable, the patient is followed up for another month after which he is subjected to the second provocation. Good results of the second provocation warrant considering the patient cured and taking him off the records.

In the case of women the provocation must consist in painting the urethra with a 1-2 per cent silver nitrate solution or Lugol's solution and the canal of the cervix uteri with a 3 per cent silver nitrate solution or Lugol's solution, an injection of 500,000,000-1,000,000,000 microbial bodies of a gonovaccine, and of drinking beer. The provocation is recommended after the end of the menses because it is then easier to find the gonococci, if there are any.

After the provocation the patient is examined over a period of 3 days, smears taken from the urethra, cervix uteri, anus and Bartholin's glands every day. If the results are favourable, the patient is not treated until the next menstruation during which smears are taken 3-4 times; a few days after menstruation the patient is subjected to another provocation.

The smears are examined during the menses and the provocation is repeated over a period of 3 menstrual cycles. Favourable results of this check-up warrant considering the patient cured and taking her off the records.

In the case of girls the provocation is made 10 days after disappearance of the gonorrheal phenomena; the provocation consists in: (1) painting the ostium vaginae and the vaginal mucosa with Lugol's solution, (2) instillation of 2-3 drops of a 0.5-1 per cent silver nitrate solution into the urethra, (3) administration of 10-15 ml of a 0.5-1 per cent silver nitrate solution into the rectum by means of a rubber syringe, and (4) intramuscular injection of 150,000,000-200,000,000 microbial bodies of a gonovaccine. This is followed by 3 days of observation during which smears are taken.

The provocation is made once a month over a period of 3 months. If the results are favourable, the girl is considered cured.

Prevention of Gonorrhea in Children's Institutions

Failure to observe the rules of sanitation and hygiene in children's institution may result in infection of children with gonorrhea. To prevent this disease, the following measures must be carried out systematically:

1. Medical examination of all newly admitted children with special attention paid to the external genitalia. On admission of children to closed children's institutions—homes for infants, all-day nurseries, boarding-schools—it is necessary to take smears from the vagina for examination. Gonorrhea-infected children are not admitted to children's institutions until cured of the disease.

2. Periodic preventive examinations of children in children's institutions. Children discovered to have gonorrhea, are immediately isolated from the other children and are subjected to treatment.

3. Medical examination of all persons hired for work in children's institutions (smears to be taken from the urethra, cervix uteri, Bartholin's glands and rectum). In dubious cases a provocation is resorted to.

The employees of children's institutions must be given a preventive examination once a month.

4. Every child must have an individual bed and towel.

5. The girls' external genitalia must be washed with running water (from a jug, cup, etc.) not in but over a basin.

6. Sponges and washcloths must be individual and must be boiled after use.

7. Each girl must have an individual chamber-pot with a special mark corresponding to the child's age (picture, number and name).

8. If there are not enough chamber-pots in the children's institution, each girl must have an individual seat which is put on the chamber-pot before use. The seats are cut out of plywood or cardboard and are covered with oil-cloth. The seats must also have individual markings.

The chamber-pots must be thoroughly disinfected every time they are used.

CONTROL OF VENEREAL AND CONTAGIOUS SKIN DISEASES IN THE U.S.S.R.

A state system of controlling venereal diseases has been elaborated and put into practice in the Soviet Union. The following are the most important elements of this system:

1. *Compulsory registration of patients with venereal and fungus diseases.* No organised or planned control of venereal and fungus diseases is possible without regular and accurate records.

In the U.S.S.R. there is a single system of keeping records of patients with venereal diseases, trichophytosis, microsporosis and favus. At whatever medical institution such a patient may apply for the first time he is immediately duly registered. At the end of the month the medical institution forwards all the records (filled-in blanks) to the district (or city) department of health which in its turn sends them to the regional (or republican) dispensary for skin and venereal diseases. These departments of health and dispensaries collect and study the morbidity data and utilise them in improving the control of venereal and fungus diseases. The departments of health are guided by the morbidity data in planning the network of medical institutions, distributing instruments, equipment and medicaments, assigning medical workers for specialisation, placing personnel, etc.

2. *Free qualified medical service for the entire population.* The socialist state strives to ensure necessary treatment for every patient. All patients are treated free of charge. The state not only provides for free medical examinations and treatment, but also allocates enormous funds for the purchase of medicaments to treat patients with venereal and skin diseases.

The extensive network of dispensaries and offices for the treatment of venereal and skin diseases has made medical aid to patients with venereal, fungus and other skin diseases really accessible to everybody. In rural communities the treatment of venereal, fungus and other skin diseases is administered in district and divisional hospitals and at medical stations, which makes it possible for all patients with venereal and contagious skin diseases to receive treatment near their domicile or place of work.

· The Soviet state spares no efforts in ensuring *qualified* medical aid for all patients with venereal and skin diseases. The U.S.S.R. Ministry of Health elaborates and issues instructions and schemes on questions of treatment and prevention of venereal and contagious skin diseases. The instructions are obligatory to all medical workers.

3. *Hospitalisation of patients with contagious forms of syphilis.*
Patients with contagious forms of syphilis (primary, recent secondary, relapsing secondary and congenital infantile and early-childhood syphilis) are subject to compulsory hospitalisation. Patients with these forms of syphilis must be retained in the hospital until the end of the first course of treatment.

In rural areas such patients are either placed in rural hospitals or are referred to the nearest dispensary for skin and venereal diseases.

4. *Follow-up methods of work.* The follow-up methods of caring for patients are the distinguishing feature of the Soviet system of controlling venereal and contagious skin diseases. Every patient with a venereal or contagious skin disease applying to a medical institution is registered.

The medical institution sees to it that the patient gets regular treatment and carries the treatment to a complete cure. Patients who do not take regular treatment or evade treatment are summoned to the medical institution where they are explained the importance of accurately carrying out all of the physician's prescriptions.

The follow-up methods of work are also aimed at *revealing the source of infection* and making the infected person or persons take treatment. It is no less important to make sure whether or not the patient has infected any of his associates. For this purpose all members of the patient's family, as well as all other persons who may have had sexual or close day-to-day contact with the patient are examined.

A venereal disease may run a concealed course and during the examination the disease may be in the incubation or latent period. Examination of the possible sources of infection must therefore be repeated several times over a period of 2-3 months.

Upon discovery of a venereal or contagious skin disease in a children's institution all the children and all employees of the institution are examined.

5. *Active revealment of patients with venereal and fungus diseases.* Soviet medical workers do not wait until the patient with a venereal or contagious skin disease applies for medical aid. Success in the control of these diseases depends on the earliest possible revealment and treatment of patients. Active revealment of patients is a characteristic feature of the Soviet system of controlling venereal and contagious skin diseases. For this purpose 100 per cent of the pregnant women are examined and wassermannised, and all blood donors and workers of children's institutions and the food industries are given preventive examinations.

Venereological and dermatological expeditions and expert groups are sent by departments of health to areas unfavourable as regards venereal or fungus diseases. The members of these expeditions

and groups conduct mass examinations of the population, treat the discovered patients, and help the local health departments and medical institutions to organise treatment and prevention of venereal and fungus diseases.

6. *Obligatory treatment and responsibility of patients for spreading the infection.* The Soviet Government has made provisions for compulsory treatment of patients with venereal diseases who avoid treatment. Soviet law provides severe penalties for those who, aware that they have a venereal disease, infect others.

7. *Health education.* In the control of venereal diseases health education is aimed at acquainting the population with the manifestations of these diseases and the routes of their spread. It must also point out the connections between venereal diseases, lechery and alcoholism.

The medical workers of venereal and skin-disease institutions, maternity health centres, rural district and divisional hospitals and medical stations deliver lectures and give talks in clubs, at industrial enterprises, on collective farms, in hostels, recreation and reading rooms, and in reception rooms of medical institutions. Moreover, special booklets and leaflets are published, and posters and films about prevention of venereal and contagious skin diseases are made.

Special attention is devoted to health education of venereal patients. They are instructed individually and in groups; leaflets and memoranda are printed to acquaint them with the manifestations and course of venereal diseases, the necessity for regular treatment, the regimen and behaviour of venereal patients, and the legal responsibility of venereal patients for infecting others.

The work of controlling venereal and contagious skin diseases is supervised by departments of specialised medical aid of the U.S.S.R. Ministry of Health and of the ministries of health of the Union Republics. Special institutes for venereal and skin diseases have been founded in the capitals and some large cities of the Union Republics for the purpose of studying venereal and skin diseases and for scientific supervision of the control of these diseases. The departments of venereal and skin diseases of undergraduate and graduate medical institutes also take part in this work.

Republican, territorial and regional dispensaries for venereal and skin diseases treat patients with these diseases in and outside of hospitals, carry out preventive measures and exercise organisational and methodological supervision of the control of venereal and contagious skin diseases in the republics, territories and regions. Dispensary physicians travel through various districts to help the local medical workers, check up on their work and organise advanced training courses.

In cities the same role is played by city dispensaries for skin

and venereal diseases and by departments for venereal and skin diseases of polyclinics.

In rural areas patients with venereal and skin diseases are treated in departments for venereal and skin diseases of district hospitals. The same departments supervise the control of venereal and contagious skin diseases carried out by the other medical institutions of their districts. In districts devoid of offices for the treatment of venereal and skin diseases the patients with venereal and fungus diseases are treated and the preventive measures are carried out by a physician of the district hospital, by rural divisional hospitals and medical stations. In rural areas venereal and contagious skin diseases can be successfully controlled only if all medical institutions and all medical workers take part in it. Without participation of the rural divisional hospitals and medical stations it is impossible to organise regular and proper treatment of patients with latent forms of syphilis, gonorrhea and fungus diseases, and to carry out preventive measures (revealment of sources of infection, examination of the members of the family and other persons who have had contact with the patients, preventive examinations in children's institutions, examination and wassermannisation of pregnant women, etc.).

An important part in the control of venereal diseases is played by maternity health centres. The physicians of these centres examine and wassermannise pregnant women, and examine gynecological patients with inflammatory processes in the urogenital organs for the purpose of discovering and treating gonorrhea patients.

The dispensaries for skin and venereal diseases, especially in large cities, have after-work preventoriums. Persons who have had dubious sexual intercourse apply to these establishments for the purpose of preventing venereal infection.

At the prophylactic station the visitor washes his hands with soap and water, and then washes with soap and warm water his external genitalia, the region of the pubes, the perineum and thighs. After washing he rubs these parts with cotton soaked in a 1 : 4,000 mercuric chloride solution and urinates, following which he is given a urethral irrigation with a 1 : 6,000 potassium permanganate solution; if the visitor is a woman, she is also given a vaginal lavage and instillation of a 1-2 per cent silver nitrate solution into the urethra, the cervix uteri being painted with a 2 per cent silver nitrate solution. Lastly, a 33 per cent calomel ointment is rubbed into the external genitalia and the surrounding skin.

To protect the external genitalia from the contaminated underwear, the patient is given a gauze napkin and is told to change the underwear immediately upon coming home.

Personal prevention produces the best results when no more than 1-4 hours have elapsed since the sexual intercourse.

Intermediate medical personnel plays an important part in the control of venereal and contagious skin diseases. In hospitals, venereological and dermatological dispensaries, maternity health centres and at medical stations, physician's assistants, midwives and nurses administer treatment to patients with venereal and skin diseases.

The intermediate medical personnel help physicians in carrying out the more complicated procedures, in making lumbar punctures, introducing bougies, urethroscopy, cystoscopy, surgical intervention, biopsy, etc.

All medical procedures require precise execution, cleanliness and asepsis (injections, etc.).

Physician's assistants, obstetricians and nurses often have to take specimens (of the blood, urine, discharge from the urethra, cervix uteri, etc.) for laboratory analysis. The quality of these specimens and, consequently, the correct results of laboratory analysis depend on proper techniques of taking the specimens, the conscientiousness and patience of these medical workers.

The intermediate medical personnel must be familiar with the causes of venereal and contagious skin diseases, their clinical picture and course. They must understand the significance of the treatment and have knowledge of the effects of the drugs and other therapeutic agents used in the treatment.

The intermediate medical personnel must often do statistical work—keep records of patients with venereal and contagious skin diseases, help physicians in drawing up reports on the work, etc. This work is very important since successful control of these diseases is impossible without proper records of patients with these diseases.

The quality of work done by the intermediate medical personnel underlies all of the work of every medical institution, from a rural medical station to clinics for skin and venereal diseases.

The regimen of a medical institution must favour the quickest possible recovery of the patients. Considerate and thoughtful treatment of the patient and a striving to dispel his anxieties and doubts must underlie the behaviour of every member of the medical personnel.

I. Pavlov wrote that "the word is as much a conditioned stimulus as anything else". The intermediate medical personnel must remember that words may strongly impress the patient. Every talk with a physician, physician's assistant or nurse must reassure the patient, cheer him up and make him believe in recovery. Such reckless words and expressions as "the patient's condition has changed for the worse", "the treatment is not helping

any", "this disease is incurable anyway", "he will probably have a relapse", etc., must be carefully avoided since they will do the patient enormous harm if he hears them.

It is very important strictly to observe the daily routine in the hospital or clinic. Regular hours for the therapeutic procedures, physician's rounds, meals and sleep are important elements of the therapeutic protective regimen.

Strict silence must be observed in the hospital during the daytime rest hours and at night. Successful observance of a proper regimen depends primarily on the work of the intermediate medical personnel.

The role of the intermediate medical personnel is particularly important in rural areas where they are responsible for the therapeutic and preventive work.

In rural hospitals the intermediate medical personnel are often assigned the reception of outpatients in the department of skin diseases of the district hospital, prophylactic examination of children in schools and other children's institutions, examination of the members of the families of registered patients with fungus diseases, scabies, etc., and talks on collective farms on the subject of venereal and skin diseases.

The role of physician's assistants and nurses working at independent medical stations is particularly important. There they are additionally responsible for timely identification of diseases, administration of medical aid to patients with venereal and skin diseases, revealment of sources of infection, examination of the members of the patient's family and his contacts, and the work of health education.

PRESCRIPTIONS MENTIONED IN THE TEXT

For the Chapter "General Treatment of Skin Deseases"

Rp. Sol. Novocaini 0.25%
Steril. 50.0
DS. 2-10 ml intravenously

Rp. Sol. Natrii bromati 0.5% 200.0
DS. 1 tablespoonful 3 times a day

Rp. Sol. Calcii chlorati 10% 10.0
D.t.d.N. 15 in amp.
S. 5-10 ml intravenously, daily or every other
day

Rp. Sol. Natrii hyposulfurosi 10% 200.0
DS. 1 tablespoonful 3 times a day before meals

Rp. Sol. Natrii hyposulfurosi 10% 50.0
Steril.!
DS. For daily internal infusions of 10 ml

Rp. Calcii gluconici 0.5 in tabul.
D.t.d. N. 30
S. 1 pill 3 times a day

Rp. Sol. Calcii gluconici 10% 10.0 in amp.
D.t.d. N. 20
S. 1 ampule intravenously

Rp. Sol. Aminazini (Chlorpromazini) 2.5% 1.0
D.t.d. in amp. N. 50
S. 1 ampule intramuscularly twice a day (befo-
re daytime rest and before sleep)

Rp. Synthomycini 0.5
D.t.d. N. 30
S. 1 powder 3-4 times a day

Rp. Biomycini (Chlortetracyclini, Aureomycini) 0.2
D.t.d. N. 20
S. 1 powder 4-5 times a day

Rp. Tetracyclini 0.1
D.t.d. N. 30 in tabul.
S. 2 pills 4-5 times a day

Rp. Sol. Novocaini 0.25% 200.0
   DS. 1 tablespoonful 3 times a day 1 hour before
      meals

Rp. Sol. Thiamini hydrochlorici 5% 1.0
   D.t.d. N. 40 in amp.
   S. 1 ampule intramuscularly (or intra-
   venously)

Rp. Thiamini bromati 0.02-0.03
   Sacchari albi 0.25
   M.f. pulv. D.t.d. N. 30
   S. 1 powder 3 times a day, half hour before
      meals

Rp. Riboflavini 0.005-0.01
   Sacchari albi 0.25
   M.f. pulv. D.t.d. N. 30
   S. 1. powder 3 times a day half hour before
      meals

Rp. Sol. Acidi ascorbinici 5% 5.0
   D.t.d. N. 20 in amp.
   S. 5-10 ml intravenously or intramuscularly

Rp. Vitamini P 0.2-0.3
   Sacchari albi 0.2
   M.f. pulv. D.t.d. N. 20
   S. 1 powder once a day

Rp. Sol. Acidi nicotinici 1% 1.0
   D.t.d. N. 100 in amp.
   S. 2-10 ml intravenously

Rp. Sol. Acidi nicotinici 0.1
   Sacchari albi 0.2
   M.f. pulv. D.t.d. N. 30
   S. 1 powder 3 times a day after meals

Rp. Vitamini A concentrati 20.0
   DS. 10-20 drops 3 times a day after meals

Rp. Norsulfasoli 0.5
   D.t.d. N. 30 in tabul.
   S. 2 pills 3-4 times a day

Rp. Aethazoli 1.0
   D.t.d. N. 20   •
   S. 1 powder 3-4 times a day

Rp. Sulfodimezini 0.5
   D.t.d. N. 30 in tabul.
   S. 2 pills 3-4 times a day

Rp. Dimedroli 0.05
   D.t.d. N. 30
   S. 2 pills 3 times a day

Rp. Sol. Dimedroli 1% -20.0
    Steril.!
    S. 1-2 ml intramuscularly or intravenously
       1-2 times a day

Rp. Diasolini 0.1-0.2 in tabul.
    D.t.d. N. 20
    S. 1 pill 1-2 times a day after meals

Rp. Diprasini (promethazini, phenergani)
       0.025-0.05 in tabul.
    D.t.d. N. 15
    S. 1 pill 2-3 times a day

Rp. Prednisoloni 0.005 in tabul.
    D.t.d. N. 100
    S. 2 pills 4 times a day

Rp. Prednisoni 0.005 in tabul.
    D.t.d. N. 50
    S. 1 pill 3 times a day

Rp. Triamcinoloni 0.004 in tabul.
    D.t.d. N. 30
    S. 1-2 pills 2-3 times a day

Rp. Dexamethazoni 0.0015 in tabul.
    D.t.d. N. 30
    S. 1-2 pills 2-3 times a day

Rp. Sol. Natrii arsenicici 1% 1.0
    D.t.d. N. 40 in amp.
    S. 0.25-0.5-0.75-1 ml subcutaneously once a day

Rp. Liq. arsenicalis Fowleri 10.0
    T-rae Ferri pomati 15.0
    MDS. 5-15 drops 3 times a day after meals

Rp. Acidi arsenicosi 0.1
    Piperis nigri 2.5
    Extr. et pulv. Liquir. q.s. ut f. pil. N. 100
    DS. 1-3 pills 3 times a day after meals

Rp. Acidi ascorbinici 0.05 in tabul.
    DS. 2 pills 3 times a day before meals

For the Chapter on "Local Treatment of Skin Diseases"

Rp. Zinci oxydati __
    Talci veneti aa 10.0
    M.f. pulv. Powder

Rp. Dermatoli __
    Talci veneti aa 7.5
    M.f. pulv. Powder

Rp. Sol. Argenti nitrici 0.25% 300.0
DS. Lotion

Rp. Sol. Aluminii acetici 1% 300.0
DS. Lotion

Rp. Aq. Plumbi 300.0
DS. Lotion

Rp. Sol. Resorcini 1% 300.0
DS. Lotion

Rp. Sol. Rivanoli ex 1 : 1000 200.0
DS. Lotion

Rp. Sol. Furacilini ex 1 : 5000 200.0
DS. Lotion

Rp. Sol. Methylenblau ex 1 : 1000 200.0
DS. Lotion

Rp. Acidi salicylici 0.5-1.0
Spiritus vini 70° 50.0
MDS. External

Rp. Acidi carbolici 0.75
Mentholi 0.5
Spiritus vini 70° 50.0
MDS. External

Rp. Zinci oxydati __
Talci veneti aa 20.0
Glycerini   __
Aq. destill. aa 30.0
MDS. External. Shake before using

Rp. Zinci oxydati __
Talci veneti aa 15.0
Glycerini   __
Spiritus vini aa 20.0
Aq. destill. 30.0
MDS. External. Shake before using

Rp. Zinci oxydati
Talci veneti
Lanolini __
Vaselini aa 10.0
M.f. pasta. Zinc paste

Rp. Acidi salicylici 0.4
Pastae Zinci ad 20.0
MDS. Lassar's paste

Rp. Ichthyoli 0.4
Pastae Zinci ad 20.0
MDS. Paste

Rp. Olei Rusci 0.6
Pastae Zinci ad 20.0
MDS. Paste

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Rp. Naphtae naphtalani 1.5
Pastae Zinci ad 30.0
MDS. Paste

Rp. Zinci oxydati 20.0-40.0
Olei persicorum ad 100.0
MDS. External. Shake before using

Rp. Ung. Acidi salicylici 3% 30.0
DS. Ointment

Rp. Zinci oxydati 3.0
Vaselini ad 30.0
MDS. Zinc ointment

Rp. Ung. Hydrocortisoni 1% 5.0
DS. Ointment

Rp. Ung. Hydrocortisoni 2.5% 5.0
DS. Ointment

Rp. Ung. Prednisoloni 0.5% 5.0
DS. Ointment

Rp. Ung. Triamcinoloni 0.1% -5.0
DS. Ointment

For the Chapter on "Pyodermas"

Rp. Methylviolet 0.25
Spiritus vini 70° 25.0
MDS. External

Rp. Brilliantgrün 0.25
Spiritus vini 70° 25.0
MDS. External

Rp. Spiritus camphorati 5.0
Spiritus vini 70-40° 95.0
MDS. External

Rp. Synthomycini (Chloramphenicoli) 3.0
Spiritus vini 70° ad 100.0
MDS. External

Rp. Ung. Colimycini 3% 30.0
DS. Ointment

Rp. Ung. Synthomycini 3% 30.0
DS. Ointment

Rp. Ung. Biomycini (Chlortetracyclini) 3% 20.0
DS. Ointment

Rp. Emuls. Synthomycini 1% 30.0
DS. External

Rp. Ung. Tetracyclini 1% 15.0
DS. Ointment

Rp. Polimyxini 200,000 u
Levomycetini (Chlormycetini) 0.6
Lanolini 10.0
Vaselini ad 20.0
MDS. Ointment

Rp. Polimyxini 200,000 u
Tetracyclini 0.6
Vaselini ad 20.0
MDS. Ointment

Rp. Oxytetracyclini (Terramycini) 03.
Ung. Hydrocortisoni 1% ad 10.0
MDS. Ointment ("Oxycort")

Rp. Ung. Rivanoli 1% 30.0
DS. Ointment

Rp. Ung. Hydrargyri praecipitati albi 3% 30.0
DS. Ointment

Rp. Ung. Xeroformi 5% 30.0
DS. Ointment

Rp. Ung. Furacilini ex 1 : 500 30.0
DS. Ointment

Rp. Acidi borici subtilissime pulverati 3 0
Ol. Rusci 1.5
Vaselini ad 30.0
MDS. Ointment

Rp. Ung. Hydrargyri oxydati   flavi 2% 30.0
DS. Ointment

Rp. Ung. Dermatoli 5% 30.0
DS. Ointment

Rp. Ung. Sulfurati 10% 30.0
DS. Ointment

Rp. Ichthyoli puri 20.0
DS. External

Rp. Ol. Rusci 5.0
Xeroformi 3.0
Ol. Ricini ad 100.0
MDS. Vishnevsky's ointment

Rp. Argenti nitrici 0.3
Balsami peruviani (s. Balsami Shostakovsky) 3.0
Vaselini ad 30.3
MDS. Mikulicz' ointment

Rp. Acidi tannici 1.0
Brilliantgrün
Spiritus vini 96° $\overline{aa}$ 0.2
Olei Ricini 0.5
Collodii 20.0
MDS. External. Novikov's solution

For the Chapter on "Fungus Diseases of the Skin"

Rp. Acidi salicylici 12.0
Acidi lactici (Acidi benzoici) 6.0
Vaselini 82.0
MDS. Arievich's ointment

Rp. Sulfuris praecipitati 5.0
Olei Rusci 2.5
Vaselini ad 50.3
MDS. Ointment

Rp. Sol. Ichthyoli 5% 200.0
DS. Lotion

Rp. Acidi salicylici
Acidi lactici $\overline{aa}$ 0.3
Resorcini 1.5
Collodii elastic ad 30.0
MDS. External

Rp. Nitrofungini 25.0
DS. External

Rp. Nystatini 2,000,000 u
Lanolini
Vaselini $\overline{aa}$ 10.0
MDS. Nystatin ointment

Rp. Acidi salicylici 0.6
Brilliantgrün
Resorcini $\overline{aa}$ 0.3
Formalini 0.15
Spiritus vini 70° 30.0
MDS. Suteyev and Asnin solution. External

Rp. Urotropini subtilissime pulverati
D. in chart. cerat. 20.0
S. Dusting powder

Rp. Sol. Kalii jodati 2% 200.0
DS. 1 tablespoonful in milk 3 times a day
after meals

Rp. Sulfuris praecipitati
Zinci oxydati
Acidi salicylici
Acidi tannici $\overline{aa}$ 1.0
Talci veneti 16.0
MDS. Dusting powder

Rp. Riboflavini 0.01
Thiamini bromati 0.03
Acidi nicotinici 0.1
Sacchari albi 0.2
M.f. pulv. D.t.d. N. 30.0
S. 1 powder 3 times a day after meals

Rp. Sol. Methylviolet 1% 100.0
DS. For lubrication

Rp. Sol. Gentianviolet 2% 100.0
DS. For lubrication

Rp. Natrii biborici 3.0
Glycerini ad 30.0
MDS. For lubrication

Rp. Nystatini 500,000 u in tabul.
D.t.d. N. 100
S. 2 pills 5 times a day

Rp. Ung. Undecyni 30.0
DS. Ointment

Rp. Ung. Amycazoli 5% 30.0
DS. Rub into affected parts

Rp. Griseofulvini 0.25 in tabul.
D.t.d. N. 100
S. 1-2 pills 2-4 times a day

**For the Chapters on "Tuberculosis of the Skin"
and "Lupus Erythematosus"**

Rp. Phthivazidi 0.25
D.t.d. N. 60
S. 1 powder 4 times a day

Rp. Streptomycini 0.5
D.t.d. N. 10
S. For intramuscular injections, once a day

Rp. Dihydrostreptomycini 1.0
D.t.d. N. 20
S. For intramuscular injections (0.5-1.0 once a day)

Rp. Vitamini D_2 15.0
DS. 50,000 I.U. twice a day after meals

Rp. Acidi paraaminosalicylici 2.0
D.t.d. N. 100
S. 1 powder 4-6 times a day

Rp. Resochini 0.25
D.t.d. N. 20 in tabul.
S. Half a pill 2-4 times a day

Rp. Acrichini 0.1
D.t.d. N. 30 in tabul.
S. 1 powder 3 times a day before meals

Rp. Vitamini B$_{12}$ 30 γ
D.t.d. N. 30 in amp.
S. 3 ampules intramuscularly daily or every
other day

Rp. Acidi paraaminosalicylici 4.5
Pastae Zinci ad 30.0
MDS. Daytime (protective) ointment (for
lupus erythematosus)

Rp. Acidi paraaminobenzoici 0.8
Lanolini ad 20.0
MDS. Daytime (protective) ointment (for lupus
erythematosus)

Rp. Saloli
Acidi tannici
Chinini sulfurici $\overline{aa}$ 3.0
Lanolini ad 30.0
MDS. Daytime (protective) ointment (for lupus
erythematosus)

Rp. Ichthyoli 1.5
Sulfuris praecipitati
Acidi borici subtilissime pulverati $\overline{aa}$ 1.0
Acidi salicylici 0.6
Vaselini ad 30.0
MDS. Ointment

For the Chapter on ‼Parasitic Diseases of the Skin"

Rp. Sol. Natrii hyposulfurosi 60% 300.0
DS. External, solution No. 1

Rp. Sol. Acidi hydrochlorici concentrati 6% 300.0
(or Sol. Acidi hydrochlorici diluti 20% 300.0)
DS. External, solution No. 2

Rp. Sulfuris praecipitati
Ol. Rusci $\overline{aa}$ 15.0
Cretae albae 10.0
Saponis viridis
Vaselini $\overline{aa}$ 30.0
MDS. Wilkinson's ointment

Rp. Ung. sulfurati 30% 100.0
DS. Ointment. Rub into the skin

Rp. Ung. Wilkinsoni
Ung. Zinci $\overline{aa}$ 50.0
MDS. Ointment. Rub into the skin

Rp. Hydrargyri bichlorati corrosivi 0.3
Aceti communis 100.0
MDS. External (for pediculosis pubis)

Rp. Acidi salicylici 0.4
Pastae Zinci ad 20.0
MDS. Lassar's paste

Rp. Sol. Dimedroli 1% 30.0
Steril.!
DS. 1 ml 1-2 times a day intramuscularly

Rp. Sol. Pyridoxini
(Vitamini B_6) 5% 1.0
Steril.!
D.t.d. N. 15 in amp.
S. 1 ml intramuscularly

Rp. Aq. Plumbi 200.0
DS. Lotion

Rp. Zinci oxydati 30.0
Ol. persicorum 70.0
MDS. Zinc oil

Rp. Naphtae naphtalani 1.5
Pastae Zinci ad 30.0
MDS. Paste

Rp. Ol. Cadini 0.4
Pastae Zinci ad 20.0
MDS. Paste

Rp. Sol. Argenti nitrici 0.25% 200.0
DS. Lotion

Rp. Ichthyoli 0.6
Zinci oxydati _
Talci veneti aa 7.5
Vaselini ad 30.0
MDS. Paste

Rp. Ol. Rusci 3.0
Pastae Zinci ad 30.0
MDS. Paste

Rp. Naphtae naphtalani 15.0
Zinci oxydati _
Talci veneti aa 7.5
MDS. Paste

Rp. Acidi carbolici 1.5
Mentholi 1.0
Spiritus vini 70° 100.0
MDS. Rubdown

Rp. Ung. Anaesthesini 5% 30.0
DS. Ointment

Rp. Ung. Dimedroli 3% 30.0
DS. Ointment

Rp. Ung. Hydrocortisoni 1%-5.0
DS. Ointment

Rp. Ung. Prednisoloni 0.5%-5.0
DS. Ointment

Rp. Ol. Cadini 0.1
Ung. Hydrocortisoni 2.5%-5.0
MDS. Ointment

Rp. Ol. Lithantracis 0.5
Ung. Prednisoloni 0.5%-5.0
MDS. Ointment

Rp. Sol. Ephedrini hydrochlorici 5% 1.0
Sterilis!
D.t.d. N. 10 in amp.
S. 1 ampule intramuscularly twice a day

Rp. Ephedrini hydrochlorici 0.025
D.t.d. N. 15 in tabul.
S. 1 pill 3 times a day before meals

For the Chapters on "Psoriasis", "Lichen Ruber Planus", and "Diseases of the Cutaneous Glands"

Rp. Psoriasini 100.0
DS. Ointment

Rp. Acidi salicylici 2.5
Olei Rusci 5.0
Sulfuris praecipitati 7.5
Vaselini 35.0
MDS. Ointment

Rp. Olei Rusci 3.0
Spiritus vini 96° 27.0
MDS. External. For lubrication

Rp. Sulfuris depurati 0.5
D.t.d. N. 20
S. 1 powder 3 times a day

Rp. Sulfuris praecipitati 2.5
Resorcini 1.0
Spiritus vini 96° ad 50.0
MDS. External (for seborrhea oleosa)

Rp. Sulfuris praecipitati 1.5
Ol. Ricini 2.5
Acidi salicylici 1.0
Spiritus vini 96° ad 50.0
MDS. External (for seborrhea sicca)

Rp. Urotropini subtilissime pulverati 20.0
DS. Dusting powder

Rp. Formalini 1.5
Spiritus vini 70° 50.0
MDS. External

Rp. Acidi borici subtilissime pulverati 15.0
DS. Dusting powder

Rp. Acidi salicylici 1.0
Acidi borici subtilissime pulverati 9.0
Talci veneti 5.0
MDS. Dusting powder

Rp. Pastae Sulseni 50.0
DS. By physician's prescription

Rp. Saponis Sulseni 100.0
DS. By physician's prescription

For the Chapter on "Syphilis"

Rp. Penicillini 600,000 u
DS. For intramuscular injections
of 50,000 u every 3 hours

Rp. Ecmonovocillini 7,200,000 u
DS. For intramuscular injections
of 600,000 u once a day

Rp. Novarsenoli 0.45
D.t.d. N. 25 in amp.
S. For intravenous injections

Rp. Myarsenoli 0.3
D.t.d. N. 25 in amp.
S. For intramuscular injections

Rp. Osarsoli 0.25
D. t. d. N. 20 in tabul.
S. 2 pills twice a day, one hour before meals

Rp. Sol. Glucosae 40% 10.0 in amp.
DS. For intravenous infusions,
10 ml each

Rp. Bijochinoli 100.0
DS. For intramuscular injections.
2-3 ml after heating and shaking

Rp. Bismoveroli 50.0
DS. For intramuscular injections
of 1 ml 2-3 times a week

Rp. Pentabismoli 100.0
DS. For intramuscular injections
of 2 ml every other day

Rp. Sol. Hydrargyri cyanati 2% 20.0
Steril.!
DS. For intramuscular injections
of 1 ml every other day

Rp. Hydrargyri salicylici 3.0
Ol. Persicorum 27.0
Steril.!
DS. For intramuscular injections
of 0.5-1 ml after heating and shaking

Rp. Ung. Hydrargyri cinerei 4.0
in chart. cerat.
D.t.d. N. 40
S. External. For inunction

Rp. Sol. Kalii jodati 2-7% 200.0
DS. 1 tablespoonful in milk 3 times
a day after meals

Rp. Sulfuris puri 0.4
Ol. Persicorum 20.0
Steril.!
MDS. For intramuscular injections

For the Chapters on "Chancroid" and "Gonorrhea"

Rp. Streptocidi albi subtilissime pulverati 20.0
DS. External

Rp. Sol. Argenti nitrici 1% 25.0
DS. For instillations into the urethra

Rp. Sol. Protargoli 3% 500.0
DS. For vaginal lavage

Rp. Jodi puri 1.0
Kalii jodati 2.0
Aq. destill. 100.0
MDS. External. For painting the urethra
and the canal of the cervix uteri

Supplement 2

PARAFFIN AND OZOCERITE TREATMENT OF PATIENTS
WITH SKIN DISEASES

Paraffin dissolved in a water bath and heated to 45°C is applied to the skin with a brush or gauze swab. The first layer of paraffin quickly congeals and is then covered with gauze or linen napkins soaked in paraffin heated to 50-60°C. The total thickness of the napkins with paraffin must reach 1-2 cm. The paraffin layer is covered with a piece of dry linen and then the whole thing is wrapped up in a heavy layer of cotton, woolen cloth or blanket. The paraffin is removed in 40-60 minutes after which the part is again covered for 1 hour with a dry dressing of cotton, woolen cloth, etc.

Ozocerite is heated in a water bath at first to 40-45°C. The gauze pack made of 15 layers of gauze is immersed in molten ozocerite and is applied to the skin. Then the ozocerite is heated to 60-80°C and another similar ozocerite-impregnated gauze pack is placed over the first one.

The ozocerite packs are covered with cotton, woolen cloth or blanket and are left on for 1 hour. After removal of the ozocerite the affected part is warmly covered with cotton or blanket for another hour.

Supplement 3

APPLICATION OF PURE ICHTHYOL IN THE TREATMENT
OF PATIENTS WITH DEEP PYODERMAS
("ICHTHYOL CAKE" METHOD)

The inflammatory focus is coated with a heavy layer of ichthyol which is covered with a thin layer of cotton of the same size as the part coated with ichthyol. Within 10-15 minutes the ichthyol impregnates the cotton, dries and the cake firmly adheres to the skin. No additional dressing over the ichthyol cake (if the latter is properly applied) is required. To remove the cake, a piece of cotton moistened with warm water is applied to it for 1-2 minutes before dressing the part, and the cake readily comes off. After opening of the node ichthyol is applied for another day or two, but only to the peripheral part of the infiltrate. The central part, where the node has opened, is not covered with ichthyol; only a piece of sterile gauze is applied to facilitate the outflow of the pus.

Supplement 4

I. PAIKIN'S HAND-WASHING METHOD FOR PREVENTION
OF PYODERMA

1. Paikin's hand-washing method for prevention of pyoderma has been proposed for workers of the peat industry where its use has made it possible to reduce the incidence of this disease. It has also proved useful for certain workers of the timber industry, agriculture and various industrial enterprises. The method is employed when the conditions of work are conducive to a dry and cracked skin, hyperkeratosis, callosity and minor injuries.

After work the hands are washed with soap and water. Then 2 litres of warm water are poured into a clean aluminium, zinc-coated or enamel basin and 5 ml of ammonia water is added. The hands are immersed for 5 minutes in the resultant 0.25 per cent warm ammonia water solution, after which they are carefully dried by patting with a towel and are coated with vaseline.

Supplement 5

COMPOSITION OF V. RAKHMANOV'S WASHING PASTE

Laundry soap	0.5 kg
Common clay	1.5 kg
River sand	250 g
Kerosene	0.25 l
Sulfuric acid 65-66 per cent	75 ml
(or vitriol oil 92-93 per cent)	50 ml

APPLICATION OF AN ADHESIVE CAP IN FUNGUS DISEASES OF THE HÂIRY PART OF THE HEAD

Adhesive caps may be used in the treatment of outpatients with fungus diseases of the hairy part of the head whenever there is a danger that the people surrounding the patient may become infected or the patient cannot for some reason be hospitalised.

The zinc-gelatin cap proposed by E. Yoffe is most commonly used.

To make this cap, 30 g of zinc oxide is mixed with 60 g of glycerin and pounded in a mortar, and 30 g of gelatin is dissolved in 90 ml of water which is heated until the gelatin is completely dissolved. The hot gelatin solution is added to the mixture of zinc oxide and glycerin and is thoroughly mixed with it. On cooling the new mixture forms a dense, hard mass—zinc-gelatin glue. A piece of the hard glue is put in a jar or bowl which is placed in hot water where the glue liquefies. Immediately after exposure to roentgen rays the child's head is coated with a thick layer of this liquid mass and bandaged in the form of a hippocratic cap. The bandage is covered with another layer of the glue. As the glue dries the head becomes covered with a closely fitting cap which prevents dispersion of the shed hair and cutaneous scales.

The cap is cut with a scissors along the midline and removed 18 days after exposure to roentgen rays. It may not be applied until such phenomena as pediculosis, secondary pyoderma, exudation and crusts on the hairy part of the head have been eliminated.

Experience has shown the zinc-gelatin cap to be very useful in the treatment of outpatients with fungus diseases.

METHOD OF APPLICATION OF ARIEVICH AND LEBEDEV'S THALLIUM PLASTER FOR EPILATION OF THE HAIRY PART OF THE HEÂD IN FUNGUS DISEASES

The thallium acetate contained in the plaster is a toxic substance, for which reason its dose must be precise; the permissible dose is 0.013 g per 1 kg of the child's weight. For example, for a child weighing 11 kg the permissible amount of this substance is $0.013 g \times 11 = 0.143$ g. This amount corresponds to 4.8 g of a 3 per cent thallium plaster ($0.143 : 0.03 = 4.77$) or 2.9 g of a 5 per cent plaster ($0.143 : 0.05 = 2.86$). A continuous, thin layer of the indicated amount of the plaster mass is applied with a metal spatula to the focus of affection and an area of 1 cm all around the focus. Before this is done the hair on the focus and within a radius of 5-8 cm is shaved off, and the hair on the rest of the head is cut short. The plaster mass is covered with strips of adhesive plaster and is left on for 18-20 days (Fig. 163). During this period the rest of the surface of the head is rubbed down with 2 per cent salicylic alcohol or is lightly inuncted with Wilkinson's ointment. After removal of the thallium plaster the hair and its fragments are easily removed with an epilating forceps. The foci are painted with a 2-5 per cent iodine tincture every morning and are inuncted with Wilkinson's ointment every evening. Such treatment with repeated epilation with a forceps is administered over a period of 5-6 weeks, a lactosalicylic ointment being applied to the foci of affection by Arievich's method once every 10 days.

METHODS OF TREATING ONYCHOMYCOSES

Treatment onychomycosis patients with keratolytic and fungicidal plasters. Keratolytic and fungicidal plasters have been proposed for the treatment of onychomycosis patients by A. Arievich and B. Lebedev.

The following keratolytic, i.e., loosening the horny substance of the nail, plasters are used: ureal plaster (contains 20 per cent urea on a plaster base), trichloroacetic plaster (10 per cent trichloroacetic acid) and salicylic plaster (50 per cent salicylic acid). The affected nail plate is softened in a hot soap and soda bath (2-3 teaspoonfuls of sodium bicarbonate per glassful of water) after which its superficial layer is scraped off with a scalpel or a safety razor blade. Then the proximal and side nail folds are covered with thin strips
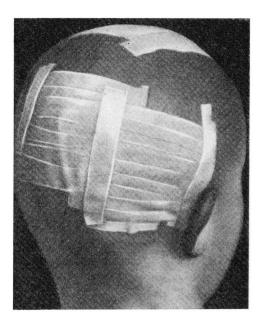
Fig. 163. Foci of fungus infection covered with thallium plaster and fastened with strips of adhesive plaster (from A. Arievich and Z. Stepanishcheva)

of adhesive plaster for protection against the irritating action of the keratolytic plaster. After this a thick layer of keratolytic plaster is spread over the nail and the entire terminal phalanx is covered with a wide strip of adhesive plaster. The dressing is changed in 2 days. The change of dressings is repeated 2-5 times and more, depending on how much the horny substance of the nail has loosened. At every change of the dressing the softened horny masses are removed with a safety razor blade, a scalpel, manicure scissors, etc.

After complete removal of the nail fungicidal plasters, i.e., plasters containing substances with fungicidal properties, are used. The following fungicidal plasters are most commonly employed: phenol plaster (5 per cent phenol on a plaster base), iodine plaster (7.5 per cent iodine and 5 per cent potassium iodide on a plaster base), betanaphthol plaster (5 per cent betanaphthol and 5 per cent salicylic acid), thymol plaster (5 per cent thymol and 5 per cent salicylic acid), and pyrogallic plaster (20 per cent pyrogallic acid). Fungicidal plasters are applied like keratolytic plasters. Alternation of fungicidal plasters is recommended, each of them being applied 2-3 times for 48 hours. The total duration of the treatment must be at least 4-5 weeks.

One of the agents used for removing fungus-affected nails is oncholysin which contains 15 per cent barium sulfate (G. Andriasyan).

An oncholysin powder is mixed with a little water and a thick layer (up to 0.5 cm) of the resultant paste is applied to the affected nail. Every 2-3 minutes the paste is moistened by addition of a drop of water with an eye-dropper. The paste must not be allowed to get on the skin surrounding the nail. In 30-40 minutes the paste is washed off with water and the softened part of the nail is scraped off with a scalpel. The application of oncholysin is repeated for another 30-40 minutes a few times over a period of 2-3 hours until the entire nail is removed. This is followed by application of a resorcinol-lactosalicylic acid ointment compress for 6 days, the dressings being changed every 48 hours.

> Rp. Acidi salicylici
> Resorcini
> Acidi lactici aa 15.0
> Vaselini 55.0
> MDS. Ointment

The nail folds are protected against the irritating action of the ointment by strips of adhesive plaster or gauze fastened with collodion.

After removal of the ointment a dressing with a 5 per cent salicylic acid ointment is applied for a period of 2 days. The softened horny strata of the nail bed are removed with a scalpel and forceps, and the nail bed is painted with a 5 per cent iodine tincture over a period of 3 days. The entire cycle of treatment — from the resorcinol-lactosalicylic acid ointment compress to painting with an iodine tincture — is repeated 3 times.

The nails can also be removed surgically or by repeated application of Arievich's lactosalicylic acid ointment for 48 hours 3-4 times with 1-day interval. After removal of the nails the nail beds are given the same antiparasitic treatment as they are after their removal with oncholysin.

A. Araviisky's method may also be used for removing nails. According to this method the fungus-affected nails are coated with a 50 per cent potassium iodide ointment.

> Rp. Kalii jodati
> Lanolini aa 15.0
> M.f.ung.
> MDS. Ointment

The surrounding skin is protected against the ointment with a layer of zinc paste or strips of adhesive plaster. This dressing is applied over a period of 10 days and changed every day. The softened nail is scraped off with a scalpel and the following ointment is applied to the exposed nail bed for 3 days.

> Rp. Jodi puri 0.2
> Kalii jodati
> Lanolini aa 10.0
> M.f. ung.
> MDS. Ointment

This is followed by 10 days' application of a 5 per cent potassium iodide ointment. The above procedures are alternated over a period of 2.5-3 months.

Supplement 9

SKIN ALLERGY TESTS

To establish the diagnosis of allergic dermatitis and to elucidate the role of the supposed allergens in the development of allergic dermatitis, eczema and certain other skin diseases, skin tests are performed.

Flap or compress skin test. A strip of gauze folded in 4 and measuring 2×2 cm is moistened with the liquid or solution under investigation (for example, a 0.5 per cent potassium bichromate solution, a 0.25 per cent novocain solution, a streptomycin solution in the concentration that was used for injections, etc.). The strip is placed on the healthy (unaffected) skin of the flexor surface of the forearm or upper arm. A compress test may also be performed on the skin of the chest, abdomen, back, and the medial surface of the thigh. The strip of gauze is covered with a somewhat larger strip of wax-paper. The wax-paper is surmounted by a still larger piece of 2-layered gauze which is fastened with 4 strips of adhesive plaster. The application is left on for 24 hours. The results of the test are evaluated in 24 and in 48 hours. A control flap test with the solution in which the substance under investigation is dissolved (water, physiologic solution, etc.) is simultaneously performed on a symmetrical part.

If the supposed allergen is a solid substance, it is ground or pulverised, placed on a strip of gauze and applied to the skin in the above manner.

Drop skin test. If the substance under investigation is a volatile liquid or is dissolved in alcohol or other organic solvent, a drop skin test is sometimes resorted to. A drop of the liquid under investigation is applied to the skin of the flexor surface of the forearm or upper arm and is left there for several minutes—until it dries. A control drop test with the liquid that has served as the solvent for the supposed allergen is simultaneously performed on a symmetrical part. The results of this test are also evaluated in 24 and 48 hours. Positive results of the compress and drop tests are manifested in redness, edema, and eruption of papules and vesicles.

Supplement 10

PREPARATION AND TRANSPORTATION OF DRY SERUM

The blood is taken from a vein, as for a usual serologic examination, with a sterile syringe and needle washed in a sterile physiologic solution before taking the blood. Some 8-10 ml of blood is drawn into the syringe, the needle is removed and the blood is carefully drained into a sterile test tube which is then plugged with cotton. To hasten the blood clotting, the test tube is placed for half an hour in warm water ($37°C$). Then a glass rod or thin wire (annealed and then cooled) is passed around the blood clot along the walls of the test tube to separate the clot from the walls. After this the test tube is placed in a centrifuge and is centrifuged for several minutes. After the centrifuging the clot settles to the bottom of the test tube, a transparent layer of serum remaining on top of it. In the absence of a centrifuge the test tube is left in a cool place for 24 hours in order that the serum may settle out. Then 1 ml of the serum is taken with a pipet or syringe and 2 drops of 0.5 ml each are made on wax-paper, cellophane or compact writing paper. The patient's name must be inscribed in a corner of the paper. The paper with the drops is allowed to dry for 24 hours protected from dust, flies and direct sunlight. For better preservation of the serum 3 drops of a 40 per cent sugar solution are added (with an eye-dropper) to the drops of serum. In 24 hours the drops of serum dry in the form of a thin yellowish film. Then the paper with the dry serum is folded (like powder paper containing powder) and is placed in an envelope together with the necessary information—name and age of patient, and reason for blood test, for example, "recent secondary syphilis", "latent tertiary syphilis," "pregnancy", "donor", etc. The envelope must be mailed *the same day* to the nearest serologic laboratory.

A special check-up has shown that examination of the dry serum yields reliable results only if not more than 10 days have elapsed between the time the blood was taken and Wassermann test with the dry serum in the laboratory.

DOSES OF PENICILLIN FOR TREATMENT OF SYPHILITIC CHILDREN

For the treatment of syphilitic infants up to 6 months of age penicillin is prescribed in a dose of 500,000 u per 1 kg of the infant's weight, but not less than 2,000,000 u per course. It is administered in a dose of 30,000 u every 3 hours. In the beginning of the *first* course of treatment the doses of penicillin are increased gradually: 2,500 u is administered every 3 hours during the first 2-3 days, then 5,000 u for 2-3 days, 10,000 u for 2 days, 20,000 u for 2 days, and then 30,000 u every 3 hours. Infants from 6 months to 1 year of age are prescribed penicillin in a dose of 400,000 u per 1 kg of weight, but no less than 3,000,000 u per course. The preparation is administered in doses of 40,000 u every 3 hours. During the first course of treatment the doses are increased gradually: 5,000 u is administered the first day, 10,000 u the second day, 15,000 u the third day, 20,000 u the fourth day, 30,000 u the fifth day, and 40,000 u from the sixth day on every 3 hours. Children from 1 to 5 years of age are prescribed penicillin in a dose of 30,000 u per 1 kg of weight, but not less than 3,000,000 u per course of treatment. The injections are made every 3 hours in a dose of 50,000 u. The first course of treatment is begun with small doses which are gradually increased. The doses are 5,000 u the first day, 10,000 u the second day, 15,000 u the third day, 20,000 u the fourth day, 20,000 u the fifth day, and 50,000 u from the sixth day on every 3 hours. Children with late congenital syphilis (5-15 years of age) are prescribed penicillin in a dose of 200,000 u per 1 kg of body weight, but not less than 4,000,000 u per course, the injections being made every 3 hours. Children from 5 to 10 years of age are injected 60,000 u, and from 10 to 15 years of age—70,000 u. During the first course of treatment the doses are increased gradually; they are 10,000 u the first day, 15,000 u the second day, 20,000 u the third day, 30,000 u the fourth day, 40,000 u the fifth day, and 60,000-70,000 u from the sixth day on, depending on age.

The treatment with water-soluble penicillin must be administered in the hospital.

Beginning with the second course, and in all subsequent courses of treatment, water-soluble penicillin may be replaced by ecmonovocillin, bicillin-1 or bicillin-3.

The daily dose of ecmonovocillin, appropriate for the age and weight of the child, is divided in half and is administered twice a day at 12-hour intervals.

Bicillin-1 and bicillin-3 are administered in single daily doses of 300,000 u, the course dose corresponding to the age and weight of the child. Children past 3 years of age, who tolerate the treatment well, may be administered 600,000 u of either of these preparations once in 2 days. Children past 7 years of age are given the first injections of these preparations in a dose of 300,000 u once a day and later 600,000 u once in 2 days. If these children tolerate bicillin treatment well, the single dose may be increased to 1,200,000 u administered once in 5 days, half of the dose being injected into each buttock.

DOSES OF ARSPHENAMINE PREPARATIONS AND OF OSARSOL (ACETARSONE) FOR TREATING SYPHILITIC CHILDREN

Children under 1 year of age are administered novarsenol (neoarsphenamine) and myarsenol (sulfarsphenamine) in a dose of 0.01-0.02 g per 1 kg of weight. The doses are gradually increased, the first injection being made in

a dose of 0.01 g per 1 kg of weight, the second injection—0.015 g and the third and subsequent injections—0.02 g per 1 kg of weight.

The injections are made once in 5 days. The single doses for infants up to 6 months of age must be 0.03-0.15 g depending on the body weight, the dose for the course totalling 0.8-1 g of either preparation. Infants 6-12 months of age are given somewhat larger doses: 0.05-0.15 g, or 1-1.25 g per course of treatment.

Children 1-3 years of age are prescribed these preparations in a dose of 0.01-0.015 g per 1 kg of weight. The single doses are 0.05-0.2 g once in 5 days, the dose for the course of treatment amounting to 1.5-2 g.

Children 3-5 years of age are given these preparations in a dose of 0.01-0.015 g per 1 kg of weight. The single doses are 0.1-0.25 g once in 5 days, 2-2.5 g being prescribed for the course of treatment.

Administration of neoarsphenamine and sulfarsphenamine to children 5-10 years of age begins with 0.1 g, the dose being gradually increased to 0.3 g for the fifth and subsequent injections. The total dose for the course of treatment is 2.5-3 g.

The dose of these preparations for children 10-15 years of age is 0.15-0.3 once in 5 days, 3-3.5 g for the course of treatment.

Acetarsol is administered to children in 5-day cycles with 3-day intervals according to the following scheme:

Child's age	Single dose	Daily dose	Per course
	in grams		
1-6 months	0.06	0.12	4
6-12 "	0.12	0.24	6
1-2 years	0.14-0.2	0.3-0.4	8
2-5 "	0.25	0.5	10
5-8 "	0.25	0.5	15
8-12 "	0.25-0.5	0.5-0.75	20
12-16 "	0.25-0.5	0.75	25

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CONTRAINDICATIONS FOR USE OF ARSPHENAMINE AND ACETARSONE

The absolute contraindications are:

(1) individual intolerance of arsphenamine preparations—neoarsphenamine, sulfarsphenamine and acetarsol;

(2) acute gastrointestinal diseases, gastric or duodenal ulcer in the stage of aggravation;

(3) severe nonsyphilitic diseases of the liver;

(4) severe nonsyphilitic diseases of the kidneys;

(5) severe nonsyphilitic diseases of the central nervous system;

(6) diffuse, acute inflammatory diseases of the skin;

(7) diabetes unamenable to dietetic treatment;

(8) heart diseases in the stage of decompensation, stable rhythm disturbances, clearly marked forms of hypertensive vascular disease;

(9) hemorrhagic diathesis;

(10) severe forms of pulmonary tuberculosis, especially with hemoptysis;

(11) clearly marked forms of exophthalmic goitre, myxedema, and Addison's disease;

(12) acute infectious diseases (angina, influenza, etc.);

(13) diseases of the visual apparatus (nonspecific iritides, iridocyclitides, keratitides, chorioretinitides, and affections of the optic nerve).

The relative contraindications are:

(1) age past 50 years;

(2) chronic intoxications (alcoholism, narcomania, lead poisoning, etc.);

(3) cardiovascular diseases (especially, myocardial);

(4) cachexia;

(5) tuberculosis of the lungs, nose, pharynx and larynx;

(6) severe form of anemia;

(7) diseases of the central nervous system accompanied by degenerative changes, epilepsy of nonsyphilitic origin;

(8) affections of the larynx with respiratory difficulties, clearly marked tonsillitides, otosclerosis;

(9) diseases of the liver and kidneys, or the presence of these diseases in the anamnesis;

(10) exophthalmic goitre, obesity;

(11) Ménière's syndrome.

SCHEMES FOR TREATING SYPHILITIC PATIENTS

1. **Treatment of syphilitic patients with penicillin and bismuth preparations.** The patients are given combined courses of treatment. A combined course begins with injections of water-soluble penicillin or ecmonovocillin. As soon as the patient has taken a course of penicillin treatment corresponding to the stage of his disease and body weight he is given injections of one of the bismuth preparations (Fig. 153).

Seronegative syphilis I. First course: penicillin (ecmonovocillin) followed by bioquinol. Interval—one month. Second course: penicillin (ecmonovocillin) followed by bismoverol, or pentabismol (monobismuth salt in neutral vegetable oil).

Seropositive syphilis I. First course: penicillin (ecmonovocillin) followed by bioquinol. Interval—one month. Second course: penicillin (ecmonovocillin) followed by pentabismol. Interval—one month. Third course: penicillin (ecmonovocillin) followed by bismoverol.

Recent syphilis II and latent syphilis II. First course: penicillin (ecmonovocillin) followed by bioquinol. Interval—one month. Second course: penicillin (ecmonovocillin) followed by pentabismol. Interval—one month. Third course: penicillin (ecmonovocillin) followed by bismoverol. Interval—one month. Fourth course: penicillin (ecmonovocillin) followed by bioquinol.

Relapsing syphilis II and early neurosyphilis. First course: penicillin (ecmonovocillin) followed by bioquinol. Interval—one month. Second course: penicillin (ecmonovocillin) followed by pentabismol. Interval—one month. Third course: penicillin (ecmonovocillin) followed by bismoverol. Interval—one month. Fourth course: penicillin (ecmonovocillin) followed by bioquinol. Interval—one month. Fifth course: penicillin (ecmonovocillin) followed by pentabismol. Interval—one month. Sixth course: penicillin (ecmonovocillin) followed by bismoverol.

Active syphilis III, latent syphilis III, latent syphilis (d'emblée), late neurosyphilis (meningovascular) and visceral syphilis. The patients with these

forms of syphilis are given the same six courses of treatment as those with relapsing syphilis II except that they must be administered perorally increasing doses of potassium iodide for 1-3 weeks before the first course of treatment.

Congenital syphilis of infants and young children (and acquired syphilis of infants and children 1-4 years of age). First course: penicillin. Interval—12-14 days. Second course: penicillin. Interval—12-14 days. Third course: penicillin. Interval—12-14 days. Fourth course: penicillin followed by bioquinol. Interval—one month. Fifth course: penicillin followed by pentabismol. Interval—one month. Sixth course: penicillin followed by bismoverol.

Late congenital syphilis and acquired syphilis of children 5-15 years of age. First course: increasing doses of potassium iodide per os for 1-3 weeks followed by penicillin. Interval—12-14 days. Second course: penicillin. Interval—12-14 days. Third course: penicillin. Interval—12-14 days. Fourth course: penicillin followed by bioquinol. Interval—one month. Fifth course: penicillin followed by pentabismol. Interval—one month. Sixth course: penicillin followed by bismoverol. Interval—one month. Seventh course: penicillin. Interval—12-14 days. Eighth course: penicillin followed by bioquinol.

During the repeated courses of treatment administered to children with congenital syphilis water-soluble penicillin may be replaced by ecmonovocillin from the second course on.

2. **Treatment of syphilitic patients with repeated course of penicillin** (Fig. 154).

Seronegative syphilis I. First course: penicillin (ecmonovocillin). Interval—2-3 weeks. Second course: penicillin (ecmonovocillin). Interval—2-3 weeks. Third course: penicillin (ecmonovocillin).

Seropositive syphilis I. First course: penicillin (ecmonovocillin). Interval—2-3 weeks. Second course: penicillin (ecmonovocillin). Interval—2-3 weeks. Third course: penicillin (ecmonovocillin). Interval—2-3 weeks. Fourth course: penicillin (ecmonovocillin).

Recent syphilis II and latent syphilis II. First course: penicillin (ecmonovocillin). Interval—2-3 weeks. Second course: penicillin (ecmonovocillin). Interval—2-3 weeks. Third course: penicillin (ecmonovocillin). Interval—2-3 weeks. Fourth course: penicillin (ecmonovocillin). Interval—2-3 weeks. Fifth course: penicillin (ecmonovocillin).

Relapsing syphilis II and early neurosyphilis. First course: penicillin (ecmonovocillin). Interval—2-3 weeks. Second course: penicillin (ecmonovocillin). Third course: penicillin (ecmonovocillin). Interval—2-3 weeks. Fourth course: penicillin (ecmonovocillin). Interval—2-3 weeks. Fifth course: penicillin (ecmonovocillin). Interval—2-3 weeks. Sixth course: penicillin (ecmonovocillin).

Active syphilis III, latent syphilis III, latent syphilis (d'emblée), late neurosyphilis and visceral syphilis. Patients with these forms of syphilis are given the same six courses of treatment as those with relapsing syphilis II except that they must be administered perorally increasing doses of iodide preparations for 1-3 weeks before the first course of treatment.

Congenital (and acquired) syphilis of infants and congenital (and acquired) syphilis of young children. First course: penicillin. Interval—12-14 days. Second course: penicillin (ecmonovocillin). Interval—12-14 days. Third course: penicillin (ecmonovocillin). Interval—12-14 days. Fourth course: penicillin (ecmonovocillin). Interval—12-14 days. Fifth course: penicillin (ecmonovocillin). Interval—12-14 days. Sixth course: penicillin (ecmonovocillin). Interval—12-14 days. Seventh course: penicillin (ecmonovocillin).

Late congenital syphilis and acquired syphilis of children 5-15 years of age. The treatment is the same as for congenital syphilis of infants except that the number of courses is increased to 8 and that the patients are given iodide preparations for 1-3 weeks before the first course of treatment.

3. **Treatment of syphilitic patients with bicillin.**

Seronegative syphilis I. First course: bicillin. Interval — 2-3 weeks. Second course: bicillin.

Seropositive syphilis I. First course: bicillin. Interval — 2-3 weeks. Second course: bicillin. Interval — 2-3 weeks. Third course: bicillin.

Recent syphilis II and latent syphilis II. First course: bicillin. Interval — 2-3 weeks. Second course: bicillin. Interval — 2-3 weeks. Third course: bicillin Interval — 2-3 weeks. Fourth course: bicillin.

Relapsing syphilis II and early neurosyphilis. First course: bicillin. Interval — 2-3 weeks. Second course: bicillin. Interval — 2-3 weeks. Third course: bicillin. Interval — 2-3 weeks. Fourth course: bicillin. Interval — 2-3 weeks. Fifth course: bicillin.

Active syphilis III, latent syphilis II, latent syphilis, late neurosyphilis and visceral syphilis. The treatment is the same as for relapsing secondary syphilis, except that the patients are given iodide preparations for 1-3 weeks before the first course of treatment.

Congenital and acquired syphilis of infants and young children. First course: penicillin. Interval — 12-14 days. Second course: bicillin. Interval — 12-14 days. Third course: bicillin. Interval — 12-14 days. Fourth course: bicillin. Interval — 12-14 days. Fifth course: bicillin. Interval — 12-14 days. Sixth course: bicillin. Interval — 12-14 days. Seventh course: bicillin.

Late congenital syphilis and acquired syphilis in children 5-15 years of age. The treatment is the same as in early congenital syphilis except that increasing doses of iodide preparations are administered to patients for 1-4 weeks before the first course.

4. **Treatment of syphilitic ptaients with bicillin and bismuth preparations.** For the treatment of patients with secondary relapsing syphilis, active tertiary and other late forms of syphilis bicillin is often effectively combined with bismuth preparations. The treatment is begun with iodide preparations given to patients for a period of 1-3 weeks.

Relapsing syphilis II, active syphilis II, latent syphilis II, latent syphilis (d'emblée), late neurosyphilis, visceral syphilis and late congenital syphilis. Potassium iodide for 1-3 weeks before the first course. First course: bicillin followed by bioquinol. Interval — one month. Second course: bicillin followed by bismoverol. Interval — one month. Third course: bicillin followed by pentabismol. Interval — one month. Fourth course: bicillin followed by bioquinol. Interval — one month. Fifth course: bicillin followed by bismoverol. Interval — one month. Sixth course: bicillin followed by pentabismol.

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TREATMENT OF PREGNANT SYPHILITIC WOMEN

Treatment of pregnant women infected with syphilis requires particular attention since it must ensure the birth of healthy offspring. This is best attained by administration of penicillin preparations which are very active against the *Treponema pallidum.* Bismuth preparations are used less frequently in the treatment of pregnant syphilitic women, while mercurials and arsphenamine preparations are not used at all.

The dose of penicillin preparations in the treatment of pregnant women corresponds to the stage of syphilis. Pregnant women with active syphilis are prescribed the following treatment: patients with seronegative syphilis I — three courses with penicillin preparations (water-soluble penicillin, ecmonovocillin, bicillin-1, bicillin-3) with intervals of 2-3 weeks between the courses. The dose of penicillin is 100,000 u per 1 kg of weight, but not less than 6,000,000 u per course. Patients with seropositive syphilis 1 are prescribed

four courses with penicillin preparations in a dose of 120,000 u per 1 kg of weight, but not less than 7,200,000 u per course, with intervals of 2-3 weeks between the courses; patients with recent syphilis II live courses with the same dose. Patients with relapsing syphilis II and active syphilis III are given six courses with penicillin preparations in a dose of 140,000 u per 1 kg of weight, but not less than 8,400,000 u per course, with intervals of 2-3 weeks between the courses. The same method is used in the treatment of pregnant women with latent seropositive syphilis (syphilis d'emblée).

In the treatment of pregnant women with active forms of syphilis and latent seropositive syphilis (syphilis d'emblée) the first course is administered with water-soluble penicillin in a hospital. To avoid an undesirable violent aggravation reaction, penicillin is administered in a dose of 20,000 u every 3 hours during the first day, 30,000 u the second day, 40,000 u the third day and 50,000 u only from the fourth day on. The second and subsequent courses of treatment may be administered to pregnant women in dispensaries, water-soluble penicillin being replaced by ecmonovocillin, bicillin-1 or bicillin-3. During the first 2 days of treatment (beginning with the second course) ecmonovocillin is administered in a dose of 300,000 u twice a day, and from the third day — if the treatment is well tolerated — in a dose of 600,000 u once a day. Administration of bicillin-1 to pregnant women begins only in the second course with an initial dose of 600,000 u. The second injection is made 3 days later also with a dose of 600,000 u, after which the preparation is administered in a dose of 1,200,000 u once in 5 days. The dose of 1,200,000 u is divided in half and is injected into both buttocks. Administration of bicillin-3 also begins only in the second course of treatment of pregnant women. The first 2 injections are made in a dose of 600,000 u with an interval of 1 day between the injections; after the first two injections the preparation is administered in a dose of 1,200,000 u once in 4 days (600,000 u into each buttock).

Even women crossed off the records after completed thorough antisyphilitic treatment must be treated during the first pregnancy again. The treatment in such cases consists of 3 courses of injections of penicillin preparations with intervals of 2-3 weeks between the courses. Must such women be treated again during subsequent pregnancies? This question has to be decided by the physician in each individual case with due regard for the individual characteristics of the patient's organism, the former treatment and the results of the preceding pregnancies.

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